UNIVERSITY OF MICHIGAN
JAN 17 1958
DENTISTRY LIBRARY

Volume 13 No. 1
January • 1958

THE AMERICAN ACADEMY OF DENTAL MEDICINE

FOUNDED 1945

COOPERATION KNOWLEDGE UNDERSTANDING

ARTICULOS SUMMARISATE IN INTERLINGUA

journal of dental medicine

OBJECTIVES OF THE AMERICAN ACADEMY OF DENTAL MEDICINE

(Quoted from the Constitution)

"To promote the study and dissemination of knowledge of the cause, prevention and control of diseases of the teeth, their supporting structures and adnexa and related subjects; and to foster and promote a better scientific understanding between the fields of dentistry and medicine."

IN THIS ISSUE

PERIPHERAL VASCULAR DISEASE AS A SOURCE OF PAIN. Edward A. Edwards 3

PAIN OF NON-ORGANIC ORIGIN. Milton Greenblatt 9

THE INTRADERMAL ASCORBIC ACID TEST PART II. A REVIEW OF HUMAN STUDIES. E. Cheraskin, J. B. Dunbar and F. H. Flynn 19

CASE REPORT: ENDODONTIA FOR CUTANEOUS LESIONS OF DENTAL ORIGIN. Morris M. Glasser 41

MEDICAL VIEWS ON ORAL PROBLEMS 45

BOOK REVIEWS 48

ACADEMY PROCEEDINGS 51

ANNOUNCEMENTS 57

JOURNAL OF DENTAL MEDICINE

VOL. 13 JANUARY, 1958 NO. 1

Editor
Irving Yudkoff, 57 West 57th Street, New York 19, N.Y.

Consultant Editor
Allan N. Arvins

Associate Editors
J. Lewis Blass
S. Leonard Rosenthal

Assistant Editors
Sanford C. Frumker
Marvin N. Okun
Alexander Soberman
Nathan Wachtel
George J. Whinston
George J. Witkin
Arthur N. Yohai

Business Manager
Harold E. Brennan, 400 Madison Avenue, New York 17, N. Y.

Publications Committee
Irving Yudkoff, B.A., D.D.S., *Chairman*
Allan N. Arvins, D.D.S.
Morton S. Biskind, M.D.
J. Lewis Blass, Ph.G., D.D.S.
Louis R. Burman, Ph.G., B.S., A.M., D.D.S.
Julius N. Obin, B.A., D.D.S.
Hayes Martin, M.D.
Samuel Charles Miller, D.D.S.
Harry Roth, B.S., D.D.S.
Sidney Sorrin, D.D.S.

* * *

The Journal of Dental Medicine is the official publication of the American Academy of Dental Medicine. It is published quarterly, in January, April, July, and October at New York, N. Y. Publication in the Journal of Dental Medicine is not an endorsement by the American Academy of Dental Medicine of opinions in any article, abstract or extract. The Editor and Publisher are not responsible for opinions expressed by authors of contributions.

Subscription rate: $4.00 Annually, $4.50 Foreign. Single copies, $1.00. Special Nutrition Issue (Sept. 1953) $1.00 per copy, special rate in quantity. Tenth Anniversary Issue (April 1956) $1.00 per copy, $5.00 for bound volume.

Copyright 1958 by the American Academy of Dental Medicine, Inc.

Preparations of Scientific Manuscripts

1. Manuscripts should be typed, double spaced, on one side of 8½ by 11 inch bond paper, allowing one inch margins.

2. The title should appear at the top of the first page, followed by the name and degrees of the author or authors. The address of the author should appear at the end of the article.

3. The name of the group before which the paper was presented, the date and place of delivery and the author's university, hospital or government affiliation, if any, should appear at the bottom of the title page.

4. Manuscripts presented for publication should: a—present new facts, describe new techniques or methods of practice or reevaluate established principles; b—contain the results of original research, or c—provide a review of facts concerning a particular subject so that the reader may deduce therefrom legitimate, important conclusions.

5. Data presented in charts and tables should be discussed but not repeated in the text.

6. A concise summary and a precise conclusion add to the value of a scientific article. The summary should state briefly the purpose of the article and the main facts presented or reviewed.

7. Bibliographic references should be in numerical sequence and correspond to the numbers placed in the text where they are mentioned.

8. An article that has been published previously under the same or a different title, or one that contains extensive excerpts from a previously published paper or book, should not be submitted for publication.

Foreign Correspondents

Argentina: Dr. Roberto Lado, Buenos Aires.
Burma: Dr. Wu Moon-Aung, Rangoon.
Brazil: Dr. Jacob Reifman, Rio de Janeiro.
Canada: Dr. Mooney Averback, Winnipeg.
Dr. Bruce Squires, Ottawa.
Colombia: Dr. Carlos de Castro, Bogota.
Cuba: Dr. Claudio Funcia Cornell, Havana.
Dominican Republic: Dr. Fernando Camino Cestero, Trujillo City.
Great Britian: Dr. A. Bryan Wade, London.
Greece: Dr. Or. A. Louridis, Athens.
Haiti: Dr. Gerald Roumain, Port-au-Prince.
India: Dr. Om Gupta, New Delhi.
Italy: Dr. Guiseppe Ceria, Turin.
Mexico: Dr. Fermin Reygadas, Mexico City.
Peru: Dr. Rolando Bernui, La Oroya.
Puerto Rico: Dr. Carlos Sosa, Santurce.
Spain: Dr. I. Saenz de la Calzada, Madrid.
Venezuela: Dr. L. J. Mota Potentini, Caracas.

DIRECTORY OF OFFICERS

President	Marcel B. Archambault, 835 St. Joseph Blvd., Montreal, Canada.
President-Elect	D. Chalmers Lyons, 420 West Michigan Avenue, Jackson, Mich.
Vice-President	Hermann Becks, 2240 Channing Way, Berkeley 4, Calif.
Secretary	George J. Witkin, 45 South Broadway, Yonkers, N. Y.
Treasurer	William M. Greenhut, 124 East 84th Street, New York 28, N. Y.
Editor	Irving Yudkoff, 57 West 57th Street, New York 19, N. Y.

CHAIRMEN OF COMMITTEES

*Constitution	G. A. Bruns, G. F. Clarke, Boston, Mass.
*Dento-Medical Relations	S. Sigmund Stahl, New York, N. Y.
*Fellowship & Honorary Membership	Don. C. Lyons, Jackson, Mich.
*Membership	Abraham Goldstein, Brooklyn, N. Y.
Planning and Finance	S. Leonard Rosenthal, Philadelphia, Pa.
*Publications	Irving Yudkoff, New York, N. Y.
Residency in Dental Medicine	Sidney Sorrin, New York, N. Y.
Sections	Samuel Turkenkopf, Newark, N. J.
*Historian	William M. Greenhut, New York, N. Y.
*Award & Endowment	Lawrence Staples, Boston, Mass.
Representative, Dental Information Bureau	Anthony Posteraro, New York, N. Y.
Alternate, Dental Information Bureau	Melvin J. Pomeranz, New York, N. Y.
*Program & Clinic—General Chairman	Don. C. Lyons, Jackson, Mich.
Chairman for 1958 Annual Meeting	George F. Clarke, Boston, Mass.
Local Chairman, 1958 Annual Meeting	Louis J. Rosen, Montreal, Canada.
Chairman for Mid-Winter Meeting 1957	Sheldon Ross, New York, N. Y.
Members Outside North America	Arthur Gold, Springfield, Mass.
Education	Harold E. Brennan, New York, N. Y.
Membership Retention	Joe Barkley, Detroit, Mich.
Ethics	J. Lewis Blass, New York, N. Y.
Keys	Louis R. Burman, New York, N. Y.
Insurance	William M. Greenhut, New York, N. Y.

DELEGATES FROM SECTIONS

Connecticut	Lester B. Weiss, New Haven, Conn.
Maryland	Irving Abramson, Baltimore, Md.
Massachusetts	John G. Goodridge, Lynn, Mass.
Montreal	Marcel B. Archambault, Montreal, Canada.
New Jersey	Abram I. Chasens, Plainfield, N. J.
New York	Harold E. Brennan, New York, N. Y.
Philadelphia	Seymour Pollock, Philadelphia, Pa.
Michigan	Edgar James, Detroit, Mich.
Ohio	Samuel Robbins, Cleveland, Ohio.
Texas	Edward T. Farris, Austin, Texas.

PAST PRESIDENTS

Samuel Charles Miller, New York, N. Y.
Sidney Sorrin, New York, N. Y.
J. Lewis Blass, New York, N. Y.
George A. Bruns, Boston, Mass.
Louis R. Burman, Bronx, N. Y.
‡Gordon R. Winter, Philadelphia, Pa.
Harold R. Gelhaar, Upper Montclair, N. J.
‡Alvin H. Berman, Baltimore, Md.
George F. Clarke, Boston, Mass.
S. Leonard Rosenthal, Philadelphia, Pa.
William M. Greenhut, New York, N. Y.

Section 31 of the Constitution: "The Board of Trustees shall consist of the elected officers of the Academy; the past presidents; the chairmen of the standing committees; and one delegate from each Section." (The President-Elect shall act as Chairman of the Board of Trustees.)

* In accordance with the Constitution.
‡ Deceased.

PERIPHERAL VASCULAR DISEASE AS A SOURCE OF PAIN*

EDWARD A. EDWARDS, M.D.†‡

Disease of the peripheral vessels resembles disease in other structures, in that pain is the most frequent presenting symptom. Unlike most disorders, however, the pain is uncommonly localized at the site of the basic pathology. This should not appear strange to a group accustomed in their daily experience to the finding of diffuse and poorly localized pain.

It is my intention to examine the patterns of pain associated with various types of peripheral disease. This will be the better understood if we inquire first into the innervation of blood vessels, and the nature of pain sensation in these structures.

SENSATION IN BLOOD VESSELS

Innervation. We may start by reaffirming that blood vessels possess the power of contraction as well as sensation, with innervation both by motor and sensory nerves. Since their muscle is of the unstriped or involuntary variety, the motor innervation is autonomic, comprising both sympathetic and parasympathetic nerves. According to current classification, these terms do not include sensory fibers. While many viscera are equally supplied by both types of autonomic fibers, sympathetic innervation is preponderant to the vessels, generally with a vaso-constrictive function. Parasympathetic supply is present, to any certainty, only in the head and neck, where it courses mainly through the facial nerve, and in the pelvis, carried by the pelvic branches of the sacral nerve. In both areas these fibers subserve the function of vasodilatation.

Sensory fibers have been amply demonstrated on blood vessels, although the innervation is much sparser than in skin. This conforms to the poor sensory supply of deep structures in general. Woollard's preparations show the fibers forming a plexus investing both an artery and its companion vein with nerves, becoming more numerous as one passes distally to the smaller vessels.[1] The branches of the plexus end in Pacinian or pressure corpuscles, or in the naked axis cylinders thought to be pain receptors. The same fibers supply both vessel walls and adjacent connective tissues. (Fig. 1.)

There is ample evidence that these sensory nerves are identical in variety with those innervating the skin, and indeed, any other tissue.[1, 2] As with other sensory nerves, the cell bodies are located in the ganglia of the posterior roots of the spinal nerves, and peripheral ganglia of cranial sensory nerves. Their axons reach the vessels either as direct branches of these spinal and cranial nerves, where they mingle with the other fibers, or by passing through the sympathetic and parasympathetic nerves and plexuses, where again they mingle with the true autonomic motor fibers of these systems. The first path through branches of spinal and cranial nerves is almost the only way in which the vessels of the limbs are innervated, except for some proximal part of the subclavian and femoral arteries, where the peri-aortic plexus extends its fibers. The second path via peri-vascular plexuses of the sympathetic and para-sympathetic, is the one mainly used by the sensory fibers for the visceral vessels in the thorax and abdomen. From the evidence available, it would appear that both pathways are used in the head and neck.

There is no doubt that in the areas mentioned, the autonomic trunks and plexuses serve as conduits for sensory fibers, many of them coming from blood vessels. What is uncertain,

* Presented before the 11th Annual Meeting of the American Academy of Dental Medicine at Boston, May 31, 1957.

† From the Department of Surgery, Peter Bent Brigham Hospital; and the Department of Anatomy, Harvard Medical School.

‡ Senior Associate in Surgery, Peter Bent Brigham Hospital; Associate Clinical Professor of Anatomy, Harvard Medical School.

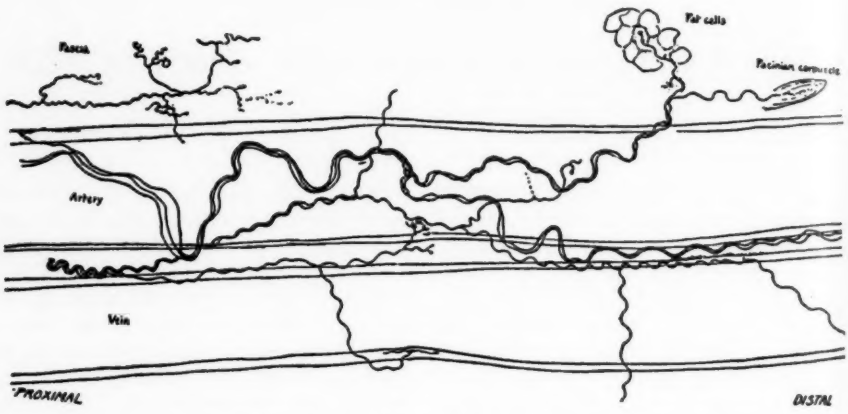

Fig. 1. Medullated nerve fibers, presumably sensory, forming an investing plexus on the tibial vessels and adjacent tissues of a cat. (From Woolard[1]).

is the exact way in which the sensory fibers extend back to their cell bodies in the sensory ganglia of spinal and cranial nerves. From both the limbs and the head and neck, there is evidence, although subject to negation by many investigators, that sensory fibers travel for considerable length in the sympathetic trunks before coursing through rami communicantes to the spinal nerves and their posterior root ganglia.[3]

PAIN ARISING IN VESSELS

It has been emphasized by Lewis and his co-workers[4] that all deep sensation, whether of visceral or non-visceral structures, is essentially similar in nature. Deep differs from superficial sensation in that:

1. It is hard to localize and is rather diffuse; that is, it is felt over a much wider area than the spot stimulated.

2. It may have superficial as well as deep reference.

3. It is accompanied by various feelings of illness.

Bazett and McGlone,[5] for example, found that pain from puncture of deep fascia, periosteum, and blood vessels, all produced a dull ache—not necessarily very painful, but difficult to endure, and associated with sweating, fainting, or nausea. Wolff and Hardy[6] produced headache in a variety of ways, including the dilatation of meningeal arteries, and traction on head vessels. Whatever the mechanism of the headache, "it was a deep aching of a diffuse nature."

What kinds of stimulation are adequate to produce blood vessel pain? Bazett and McGlone confirmed the usual impression that arterial puncture is painful, especially puncture of the smaller arteries, which conforms to Woollard's observation of a richer nerve supply to the smaller vessels. Puncture of a vein was found to be less painful than that of an artery. Anyone who has clamped an artery in an operative field under incomplete local anesthesia knows that intense pain is thus produced. It may of course be due to crushing of the peri-vascular tissue, but since the same fibers supply both vessel and this tissue, one may as well say the nerve fibers of the vessel are stimulated. That traction on vessels is painful was demonstrated by Wolff and Hardy in the head. Much of the pain of traction of a mesentery may also arise from the vessels within it.

The demonstration by the same investigators of pain production by vessel dilatation has been mentioned. They denied that vessel constriction is painful. The intravascular injection of irritant

solutions may produce great pain, particularly if the blood flow is sluggish, allowing for less dilution and more time of local action. Such injections are especially painful when accompanied by vasospasm; this may simply indicate a concomitant effect on the musculature of the vessel, or it may be that the constriction increases the pain, possibly by altering the tissues supplied by the constricted vessels.

PAIN IN PATHOLOGICAL PROCESSES

The pain engendered by disease has a dual origin in the diseased vessel, and the tissues whose circulation is altered.

Pain originating in the diseased vessel. The experiments alluded to prepare us to understand that in order to be locally painful, the pathological process must produce a considerable stress in the vessel structure. Thus dissecting aneurysm of the aorta, or rupture of a saccular aneurysm produces some of the most excruciating pain ever experienced. It is felt over the dorsal spine when the lesion is in the thoracic aorta, and over the lumbar spine and sacrum when the abdominal aorta is involved. The chronic or subacute stretching of varicose veins, which involves microscopic tearing, is also locally painful, the pain being worse on standing, when hydrostatic pressure exerts its dilating influence. A "tight" feeling of distention is characteristic of high venous pressure in conditions of venous stasis.

In distinction to these examples is the complete freedom from pain in a vessel involved in arteriosclerosis, in which the alterations do not involve violent mechanical stimulation or acute inflammation. Indeed so far as one can judge, even an acute thrombosis of a sclerotic artery causes no pain in the vessel.[7] This may be due to lack of innervation in the atheroma within which the thrombus lies. (Fig. 2.) Even in normal arteries, however, the lodgement of an embolus is not locally painful.

A similar process of bland thrombosis in a vein is usually painful, and often extremely so, the patient localizing the thrombus quite accurately. In the deep veins of the calf, this pain is initially described as a cramp or knotting up. This may indicate a change in the calf muscle.

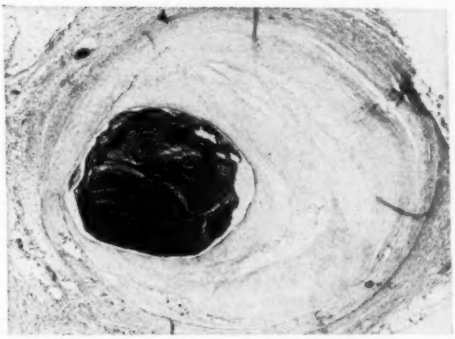

Fig. 2. Thrombosis in an arteriosclerotic artery. Note the lack of cellularity of the atheroma and the lack of inflammation. Such a process is not locally painful. (From Edwards[7]).

Later the pain is described as a deep ache. Since veins are not as richly innervated as arteries, their more acute sensation may depend on the thinner barrier separating the lumenal process from the sensitive tissues outside the vessel.

In both veins and arteries an acute inflammation, with or without thrombosis, is locally very painful indeed. In Buerger's diseases, for example, the characteristic arteritis and phlebitis are typically extremely painful; the pain extending distally through the limb, being present at rest or activity; and out of proportion to the degree of ischemia. (Fig. 3.) The spread of the inflammation to the nerve trunks, which in so many areas lie adjacent to the vessels, increases pain immeasurably. (Fig. 4.) Similar pain of vessel inflammation is noted in temporal arteritis. Other examples in the extremities are panarteritis nodosa and the angiitis of other varieties of so-called collagen disease.

PAIN ORIGINATING IN THE VASCULAR TERRITORY

Vessel pathology usually involves vascular obstruction. The resultant changes in the tissues supplied by the involved vessel are more generally productive of pain than any but the more violent changes in the vessel itself. So far as the peripheral arteries are concerned, we must therefore inquire into the pain produced by ischemia of the various tissues of the limb.

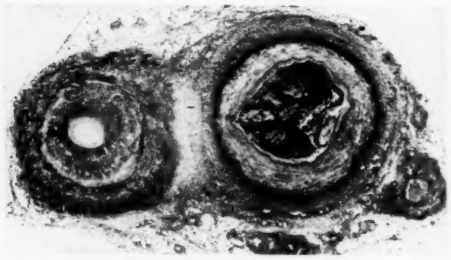

Fig. 3. Arteritis and phlebitis in Buerger's disease. A thrombus is present in the artery to the right. A violent inflammatory reaction extends throughout the walls of both vessels and beyond. Such processes are extremely painful locally.

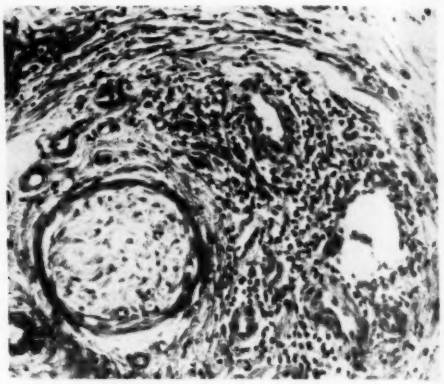

Fig. 4. Inflammation about a nerve adjacent to the vessels of Fig. 3. The nerve involvement adds greatly to the pain of this disease.

In arteriosclerosis of moderate severity, pain may be completely lacking at rest. On walking, the muscles, whose requirements are then 20 or 30 times greater than at rest, rather quickly enter into the spasm of severe fatigue—the syndrome of intermittent claudication. Characteristically, the pain is described as a cramp, usually experienced in the calf, and accompanied by a feeling of utter fatigue of the limb. It comes on after a certain distance of walking, is relieved rather promptly and completely by resting but a minute or two, and recurs on walking the same distance. The pain may be located higher or lower, depending on the arteries involved.[8] As with other deep tissues, pain originating in a muscle may extend considerably beyond the confines of that structure. With severer grades of ischemia, an ischemic neuritis may develop, with diffuse lancinating pain occurring at rest or activity. This is usually accompanied by a burning sensation in the feet, and with a moderate hypesthesia to touch and vibration sense, over a stocking distribution. In diabetes a more severe neuritis may be present, with anesthesia of the foot and absent tendon reflexes.

In extreme degrees of ischemia, pain is constant—so-called rest pain, located in the foot and toes. With such degrees of ischemia, the pressure in the distal arteries is very low, and the patient seeks relief by hanging the feet over the edge of the bed, to obtain some aid of hydrostatic pressure in increasing blood flow. Skin breakdown and infection always increase the pain. Under such conditions even the insensitive feet of diabetic neuropathy become painful.

The acute ischemia of peripheral arterial embolism or thrombosis gives rise to sudden and severe pain. Crampy pain felt in the calf or other muscles is usually the initial symptom of such an obstruction in the lower limb. It is accompanied by "pins and needles" sensation in the toes—presumably because of ischemia of the nerves. There may be distal anesthesia in a stocking distribution, with maximal pain and tenderness in the borderline between the anesthetic and sensitive tissues. When the ischemia is extreme, the muscles go into contracture or rigor, with constant severe pain. This is an ominous sign, presaging necrosis of entire muscles.

Peripheral arteriospasm per se, as in Raynaud's disease and other cold sensitivities produces a combination of numbness and pain. Lewis believed that the cooling of the tissues incident to vascular spasm or obstruction hindered the transmission of impulses subserving touch and light pressure, but had less effect on the transmission of pain impulses. Both he and others have also postulated the formation, in vasospasm, of pain producing material in the periphery. Lorente de Nó is quoted as having

demonstrated that nerve fibers when anoxic fire off repetitive, and presumably painful, stimuli.[3]

CAUSALGIA AND POST-TRAUMATIC DYSTROPHY

It is mainly because the sympathetic nerves, with which the vascular surgeon has considerable acquaintance, are involved in the mechanism of these disorders, that they should be mentioned in this discussion. Causalgia itself is an extremely severe neuralgia caused by injury (usually partial) to a large peripheral nerve. It is characterized by the same dysesthesia—that is, burning pain with diminished touch and pinprick sensation—which is also found in ischemia. The pain is felt through the hand or foot and is constant. It is increased by any touch or jarring—or even if the patient is startled. Vasodilatation may be present early, but vasoconstriction supervenes in the late stages. It is accompanied by tapering of the digits and smooth glossy skin. Sympathectomy classically gives full relief. This would seem to prove that fibers carrying pain from the extremities course through the sympathetic trunks and are thus interrupted. White and Sweet summarize the evidence against this view, and present the view of Doupe, Cullen, and Chance that peripheral nerve injury brings about an abnormal contact between sympathetic and sensory fibers which allows motor impulses travelling over the sympathetic fibers to activate the sensory pain fibers.

Post-traumatic dystrophy is often, and probably erroneously, listed as a variant of causalgia. This syndrome occurs after a variety of damage to peripheral tissues, occasioned by trauma or infection. Phlebitis of deep veins is an occasional causative disorder. Pain is the presenting symptom, often out of proportion to the severity of the injury, and lasting far beyond the time usually required for healing. The pain is not exclusively burning in nature. Vasospasm is usually present, with sweating and cyanosis. X-ray discloses a spotty decalcification of the bones of the extremity. Hysteria is often present, the term being used quite in distinction to conscious malingery. While sympathectomy may be helpful, it is by no means as constantly curative as in causalgia.

SEQUEL. PAIN AFTER AMPUTATION

The same feelings of disfigurement, disability, inferiority, and fear of further loss, which I am told patients experience after dental extractions, are felt most profoundly by patients who suffer amputation. This mood enhances the pain felt after amputation and often prevents its successful management.

In favorable cases, once the stump is healed, it is comfortable, except for such twinges as may be felt in the vicinity of any large scar. In others, however, there is pain for months or years. Only seldom is this traceable to a resectable neuroma. More often it is more diffusely felt. The mechanism of such diffuse pain is not clear, although it is certain that psychologic trauma plays a major role.

The most intractable pain is that associated with phantom limb. Phantom feeling—the sense of the continued presence of the amputated part—is probably experienced by every patient after amputation, but it tends to wear off after weeks or months. The association of pain with this persistent image is fortunately rare. The uncertainty of its cure by further amputation, by cordotomy, and even by excision of that part of the post-central gyrus serving the limb, has led to the conclusion that phantom pain is closely associated with central perception of his body image by the patient. Further, this perception of body image appears to be represented at several levels of the brain. In my experience events, such as long continued ischemic pain, which existed prior to the amputation, are effective in causing the excessive fixation of the patient on his limb, and thus provide the background for the pain. For the same reasons, a psychiatric approach is often required in its cure.

CONCLUSIONS

1. Veins and arteries receive sensory nerve supply and are sensitive to a variety of stimuli and to inflammation.

2. As with any deep structure, the pain is diffuse, hard to localize, and is accompanied by a variety of feelings of illness.

3. Pain of pathological processes is due in

part to local irritation of the vessel wall, and in part to changes of ischemia or venous distention of the tissues supplied by the diseased vessels.

Harvard Medical School,
Boston, Mass.

BIBLIOGRAPHY

1. Woollard, H. H., The innervation of blood vessels, Heart 13: 319, 1926.
2. Hinsey, J. C., Three types of fibers going to skeletal muscle and its blood vessels, Publ. Ass'n. Res. Nerv. and Ment. Dis. 9:153, 1930.
3. White, J. C. and Sweet, W. H., Pain. *Its mechanisms and neurosurgical control.* Springfield, Thomas, 1955.
4. Lewis, T., *Pain,* New York, Macmillan, 1942.
5. Bazett, H. C. and McGlone, B., Note on the pain sensations which accompany deep punctures, Brain 51: 18, 1928.
6. Wolff, H. G. and Hardy, J. D., On the nature of pain, Physiol. Rev. 27: 167, 1947.
7. Edwards, E. A., *Thrombosis in arteriosclerosis of the lower extremities,* Springfield, Thomas, 1950.
8. Edwards, E. A., Atypical manifestations of peripheral arteriosclerosis, New Eng. J. Med., 247: 627, 1952.

MORBO DE VASOS PERIPHERIC COMO FONTE DE DOLOR

Dr. med. Edward A. Edwards

SUMMARIO IN INTERLINGUA

Morbo de vasos peripheric resimila morbos in altere structuras in le facto que dolor es le plus frequente symptoma de presentation. Tamen, per contrasto con le situation in altere morbos, le dolor de morbo vascular peripheric non es communmente localisate al sito del pathologia fundamental. Iste facto non pote esser surprendente pro un gruppo de specialistas como le odontotherapeutas qui es accostumate in lor experientia diurne al phenomeno de diffuse e mal-localisate dolor.

Conclusiones.—1. Venas e arterias recipe un provision de nervos sensori e es sensibile a un varietate de stimulos e a inflammation.

2. Como in le caso de omne structuras profunde, le dolor vascular es diffuse, difficile a localisar, e accompaniate de un varietate de sensationes de morbiditate.

3. Le dolor de processos pathologic es causate in parte per un irritation local del pariete vascular e in parte per alterationes de ischemia o per distension venose del histos servite per le vasos morbide.

PAIN OF NON-ORGANIC ORIGIN*

MILTON GREENBLATT, M.D.†

Although not necessary for life, pain plays a major role in influencing our adaptation and sense of well being, and in forming and determining personality structure. The complex problem of pain and its meanings is perhaps rendered more simple by considering two aspects: (a) the perception of pain, and (b) reaction to pain.

NEURAL MECHANISMS FOR PERCEPTION OF PAIN

Pain endings have been demonstrated in the skin and other organs. Their fundamental receptors are fine medulated nerve endings which ramify rather freely in sensitive areas. These endings represent the peripheral components of neural elements whose central components find their way by dorsal horn to spinothalamic tracts or contralateral side up to thalamic nuclei, whence next order neurons make their way primarily to cerebral cortex especially in the post central area. Appreciation or consciousness of pain arises from structures above the level of the upper pons, thalamus, hypothalamus and cerebral cortex. Lesions of the upper pons will permanently abolish the appreciation of pain from peripheral stimulation. The thalamus is capable of appreciating pain and apparently provides a low order of pain differentiation by integrating pain with other modalities. From studies of lesions in man we know that thalamic pain is peculiar, distorted and associated with disagreeable feeling tones. At the highest cortical level pain sensation is highly localized in the body scheme, and colored by associated tactile and pressure sensation. At this level the state of excitation and activity of the brain influences experience of pain.

PAIN THRESHOLD AND ITS ALTERATION

We are indebted to Harold G. Wolff[1] and his collaborators for basic studies on pain and pain threshold. Using the Hardy-Wolff-Goodell apparatus which focuses a beam of light from a 1,000-watt bulb on the forehead, they have established that the pain threshold is a highly stable and uniform function in man. This may come as a surprise to those who have been impressed with their clinical observations that the reaction to pain may be influenced by a wide variety of events. It has also been shown that the threshold for tissue destruction is only about twice the threshold for pain. Thus pain is a very efficient warning that tissue may be destroyed. And when we perceive the slightest degree of pain we have learned to protect ourselves instinctively.

Pain in some respects is different from temperature sense in that pain threshold is not based on spatial summation; that is to say, by stimulating increasingly larger areas of skin with a below-threshold stimulus we do not get pain. The stimulus has to be adequate for each given area. Once adequate, increasing areas that are painfully stimulated warn the organism of increasingly more severe threats to its well being. With warmth sensation, on the other hand, the larger the area warmed at a given level, the greater the heat sensation.

The threshold of pain may be altered by various physical conditions, for example, traumatic deformation, denudement or injury of tissues may lower the threshold considerably. Sunburn, for example, may lower the threshold by 50 per cent. A rise in threshold may occur with injury of the peripheral nerve, as with an inflammatory neuritis, or from lesions of the cords, such as syringo myelia. Lesions of the internal capsure and of the thalamus raise the

* Presented at the 11th Annual Meeting of the American Academy of Dental Medicine, Boston, Massachusetts, May 30 to June 1, 1957.

† Assistant Clinical Professor of Psychiatry, Harvard Medical School; Assistant Superintendent, Massachusetts Mental Health Center, Boston.

[1] Wolff, H. G., and Goodell, H. The Relation of Attitude and Suggestion to the Perception of and Reaction to Pain. ARNMD, Chapter XXXII, 434-448, 1943.

pain threshold too, and in case of thalamic lesions, the patient has distortions of pain sensation such as burning and other parasthesias. From diseases of the post central gyrus there may be impairment of pain sensation although it is surprising at times how little the appreciation of pain is involved.

Beyond these essentially physical or pathological conditions there are pyschological factors which may influence the threshold. Many factors having to do with attention, concentration, aims, wishes and patient-doctor relationship influence the reaction to pain. It has been shown, for example, that the pain threshold is more variable if no clear instructions are given as to what the subject should observe and report, and relatively more stable if the situation is clearly structured.

The effects of distraction, auto-suggestion and hypnosis on the pain threshold have been studied under laboratory conditions. The effects may be remarkable; for example, reading a highly adventurous story increases the threshold by 16 per cent; distraction by repeating digits increases the threshold by 45 per cent; auto-suggestion ("I won't feel pain") increases the threshold by 20 per cent; shallow hypnosis by 40 per cent; and clanging a loud bell by 38 per cent.

EFFECTS OF PLACEBOS

Wolff has published studies on the effects of placebos and analgesics on a relatively non-suggestible male subject. Aspirin, .3 gms., regularly raised the pain threshold some 30 per cent. This man failed to show threshold change when a placebo (soda bicarbonate) was given, although definitive suggestion was made that an analgesic had been given. Experiments on a suggestible woman on the other hand gave somewhat different results. In her case, .9 gm. of aspirin gave a 31 per cent rise in threshold. Lactose placebo with the patient expecting aspirin gave 20 per cent rise. When the patient was told that she had been deceived and then given aspirin, no elevation of threshold occurred. This experiment perhaps suggests that if a woman has been deceived, unexpected results may be anticipated.

In patients who respond to placebos with an elevation of pain threshold, who are then informed that an inactive agent has been given, there has been a gradual descent of the elevated pain threshold to normal. Why the return to normal is not sudden is not readily explained, except perhaps in terms of slow adoption of a new psychological set and tendency for perseveration of previous response level.

It has further been shown that suggestibility, attitudes of doubt, and sleepiness alter the pain threshold. Sleepiness apparently increases the subject's willingness to believe in suggested ideas.

Thus Wolff and his collaborators have shown that a variety of psychological factors—doubt, lack of confidence, relative alertness or sleepiness, suggestibility, and belief—are all relevant factors in altering the pain threshold. There are, moreover, special conditions under which the pain reaction and perhaps threshold may be altered radically. For example, during the excitement of games or combat or sex activity, pain may readily be tolerated, even to the point of tissue damage. We are becoming increasingly familiar with indifference to pain during parturition in women who are confident in their physician, desirous of bearing children, and who are convinced both that pain will be limited and within their tolerance.

RELATION OF PERSONALITY TO ATTITUDE TOWARDS PAIN

The relation between personality and attitude to pain may be illustrated by the reaction of two young mothers, both secretaries in my department, to the pain of childbirth. Each mother has had two children; one mother has experienced the events without anaesthesia; the other with anaesthesia. Because of the extraordinary revealing contrast, I quote in some detail their feelings about the events.

The first is the mother who went through childbirth without benefit of analgesics:

"I went into this because I was curious and rather narcissistic. Anything to do with childbirth is a very important female function, if not the most important one. There were nine months of emotional involvement shared with

my spouse. One undergoes mental and biological changes. The focus of all this time is on the final birth, and we were very joyous in anticipation. I couldn't see going through an experience for nine months, being completely aware of everything, and then having to be blocked out for the finale. I felt that if I were anaesthetized, I could not be as keen as usual, then there would be unrealistic distortion. The real and unreal would be indistinguishable. To me, it is very frightening not to know which is real and which is not real. There would be a sense of incompleteness if things did not come to a real conclusion.

"Pain in this instance meant to me pleasure. This was the only time I endured this type of pain. It satisfied me to know that I could make friends with pain because there was a reward, a child, involved. It is then not as painful for a woman to accept the fact that you have to separate yourself from the child after it is born. You feel more sure, more convinced that you did give birth to the child. I felt that in the birth I was giving away something very precious to me, an act that I might want to re-experience later, and you can't relive something you are not aware of.

"How much pain? Very hard to describe. I felt it necessary to undergo a certain amount of discomfort, but I didn't focus on the pain. My curiosity of what was going on cut down on the pain. I wasn't concentrating on it. Twice I had to scream, but there was an interval of from a few seconds to a minute between severe pains, so I could adjust to it. The first sharp pain was so severe that the second didn't hurt as much.

"In some respects I feel more adequate having met the challenge, and am sorry for others who didn't select this method of childbirth. I am conscious of a certain air of superiority. Also I felt that others under the anaesthetic had no control.

"Now, about dental pain. I should want novocain if a tooth were to be extracted. I would feel that I did not initiate the pain. It was started by something else, and I wouldn't see any sense in standing that pain when novocain would allay it. In the case of pain of childbirth, I am the one who can see it through to comfort. I am the controlling agent. In the case of dental pain, the dentist is the one who relieves me."

Contrast the above with the following personal recollections of our other lady:

"Initially, I should like to state that I had been thoroughly prepared, emotionally as well as physically, for the birth of my first child. I had been happily looking forward to motherhood; had a most enjoyable pregnancy and had been perfectly able to work until my ninth month. During this entire period, I devoured a sizable amount of pertinent literature and had not hesitated to discuss necessary clarifications with my obstetrician.

"I was quite aware of the revival of natural childbirth, but somehow the testimonials on this topic proved to be nothing more than interesting reading. It was definitely not an experience I wanted to go through, nor did I possess the curiosity to try a sample. Fear was not a factor . . . I simply felt that I did not want unnecessary discomfort, and the 'thrill' of seeing my baby born was not enough to sway me, especially since I fail to look upon this moment as 'thrilling.' I knew that with my complete cooperation, the minimum amount of sedation would suffice but I believed that this minimum amount would be most thankfully received—and it sure was!

"My contractions were nothing to speak of —very mild but consistent—and there was little discomfort involved during labor. I was given a mild sedative that had not even caused me to be confined to bed, but during the last hour, I was elated when my doctor said it was time for me to go to sleep.

"I gave birth to a son two years later. This delivery was extremely fast. Somehow, this time I was far more uncomfortable and the session in the delivery room seemed endless and most unpleasant. My anaesthetist assisted me with each contraction and how anxiously I waited for that one good dose that would send me off to dreamland. The labor period had been too brief—the baby was too large and emerging too quickly—and I can remember being pretty unhappy in that delivery room. I can remember

saying, 'To hell with this natural childbirth—put me to sleep, damn you!' (My poor doctor.) Finally, my wish was granted and my son was born.

"In both cases, there were no after effects from the anaesthesia. I remained asleep only a short time after delivery and was both cheerful and hungry. In the future, if I have more children, I would prefer sleeping through the entire ordeal, labor et al, since my experience wasn't one that I would want to repeat."

These two cases show a vivid contrast in attitude towards pain and childbirth which, in turn, reflects totally different personality types. In the first instance we are dealing with a woman for whom strength and mastery of pain and the challenge of childbirth are very important. She is also the kind of person who finds it possible to obtain a special masochistic pleasure from the pain of childbirth. The feeling of superiority from having successfully met the challenge is a source of major gratification to this woman. She further highlights the extremely personal meaning of the whole process of childbirth versus the impersonal meaning of dental pain.

In the sencond instance, the problem of challenge and mastery is not even mentioned. This woman regards testimonials on natural childbirth as "interesting reading material" but definitely not an experience she chooses to undergo. Her point of view is, "Why face unnecessary discomfort."

The attitude toward pain of childbirth in each instance is probably an enduring one; one girl states that if she were to have more children she would want to face pain bravely, the other wants to avoid unnecessary discomfort.

HYPNOSIS AND PAIN

Now let us consider the problem of relationship of hypnosis to pain endurance and reaction, and parenthetically, the relationship of hypnosis to the practice of dentistry. The second topic takes us a little far afield but cannot be readily separated from the first, especially before such an audience.

I will discuss the problem of hypnosis in the form of an interview which I held with one of my research colleagues, Dr. Martin Orne, an acknowledged expert, who for some ten years has interested himself in the research and clinical aspects of hypnosis.

Q. Does hypnosis increase pain tolerance?

A. Definitely yes. Sometimes the pain endurance increases very greatly. Much depends on the success in achieving trance and the depth of trance. We must remember that some 5 to 15 per cent of subjects cannot be hypnotized and that something like 15 to 20 per cent go readily into deep trance. In between these two extremes there is a large middle group that under present techniques goes into various levels of trance from light to medium.

Q. Does pain tolerance vary in these groups?

A. The group that goes into deep trance is adequately hypnotized so that it is possible to do almost any operative procedure including nerve extractions without chemical anaesthesia. For the large middle group, about one half of this group can tolerate mildly painful procedures such as drilling although it would not be possible to manipulate nerves. Some degree of analgesia is obtained or one may need much less novocain. The rest of this group may not have a great deal of analgesic effect but the trance quiets them greatly, gives them reassurance and anxiety may be dramatically reduced, although anaesthesia is light.

Q. How do you explain the increased pain tolerance under the hypnotic situation?

A. Recent studies (from our laboratory and others) indicate a far greater role for the factor of motivation during hypnosis than formerly suspected; that is, hypnosis with its great increase in suggestibility motivates the patient to do what the hypnotist wants him to do and feel. It has been shown that tolerance to pain and other kinds of discomfort may be greatly increased if patients are similarly motivated during wakefulness. The best kinds of rewards are ego rewards, that is, the feeling that one has faced the challenge and met it successfully.

Thus if a man is asked to tolerate pain in the presence of a pretty woman he is apt to achieve a high level of pain tolerance, and if

in addition the factor of his masculinity is brought into the picture, that is, he is equal or better than the next man for tolerating pain, his pain tolerance level goes up still higher.

However, in addition to the motivation factor as such we feel there may be something very special in hypnosis perhaps beyond both suggestibility and motivation that may explain the analgesia.

Q. Do you recommend performing dental procedures in the post-hypnotic phase, having suggested to the subject that he will have no pain when he awakens?

A. Not as a rule. Analgesia tends to be most efficacious during hypnosis and, in addition, there is the added advantage of amnesia.

Q. Does one get behaviorial and physiological reactions to pain stimulus under hypnosis?

A. Yes. The patient may even flinch during the dental operation yet appears to forget it afterwards. The implication is that there is definite central nervous response but without the remembered experience of pain.

Q. Does one also get physiological changes?

A. Yes. Sears in 1932 showed that flinching and GSR persist. We found in 1949 that GSR and heart rate responses are present even in the absence of flinching. Electroencephalographic studies during hypnosis show that light and sound "not heard" by the subject alter the EEG pattern in the expected manner.

Q. How much depends on the hypnotist's technique?

A. A great deal. Some dentists who use hypnosis I'm sure are not using it in the optimal way.

Q. How do you mean?

A. First, it is important not to rush patients. Dentists seem to be in a hurry. It is not that the patient necessarily needs more time, he merely needs the feeling of not being in a hurry. This mostly means that the dentist should not himself feel in a hurry and thus impart this attitude to the patient. Secondly, dentists are likely to view hypnosis like novocain or gas, something they do *to* the patient rather than *with* the patient. It is important that the patient have a sense of participation in a cooperative enterprise; thus he will go into trance more readily. Thirdly, hypnosis is so often related to a sense of power or dominance; a dentist may feel he is in a competitive position and try to dominate the patient. The patient naturally does not wish to be overpowered. He may resist going into deep trance or react badly to it.

Q. You imply that the best results are obtained in deep trance. Could you define that state?

A. I would describe it as a state of apparent passivity in which suggestion can elicit all classical phenomena such as hallucinations, anaesthesia, age regression, amnesia and response to post-hypnotic requests. In this state reality testing is limited and the subject tends to be undisturbed by logical ambiguities. To a naive observer, he will look asleep.

Q. Do you think that hypnosis has promise as a therapeutic tool?

A. Indeed I do, but I feel that training is a very important problem. I think it would not be advisable for dentists merely to read up, go to a few lectures and try the technique. On the other hand, I feel that the use of hypnosis should not be relegated to a consultant hypnotist who would come in and help the dentists in his work with the patient. If hypnosis is to be optimally used, the technique should be developed by the dentist so that he can work within the developing dentist-patient relationship.

Q. To what extent are the dentists interested in hypnosis as a technique in dentistry?

A. There is a great deal of current interest among the dental profession. Unfortunately, there has been a great problem of obtaining adequate training. Some dentists have taken courses with stage hypnotists for want of trained teachers and have unfortunately viewed hypnosis as a separate field apart from psychology. Recently some of the dental schools have begun to offer instruction in the technique of hypnosis. While the courses offered must be developed further, this has been a very signifi-

cant step in the direction of taking hypnosis out of the hands of incompetently trained individuals.

Q. Do you see the study of hypnosis as being an important element of the undergraduate curriculum in the future?

A. It is hard to predict. I am convinced that hypnosis should not be taught per se but should be part of general instruction in psychology and psychodynamics. Hypnosis must be used within a framework of sophisticated understanding of the doctor-patient relationship. Probably an intensive course in psychodynamics should be introduced at the undergraduate level in the dental curriculum much as has taken place in the last ten years in undergraduate medical training. Special instruction in hypnotic technique should perhaps come later for individuals who are interested and adequately prepared. I feel there is no question that this trend would increase the interest of the dentist in the patient as a person and eventually increase understanding, confidence and mutual acceptance.

Q. What are the dangers in the dental use of hypnosis?

A. The dangers include the possibility of evoking a psychosis in a susceptible person or the production of additional symptoms in a neurotic subject even though some others may be removed. Perhaps the most common danger is that hypnosis may aggravate problems of transference and counter-transference, and as such, hypnosis should not be used for patients undergoing psychotherapy or psychoanalysis unless there is thorough consultation with the patient's psychiatrist.

There are reactions to hypnosis such as a headache and "dopey" feeling which must be understood, the latter often disguising hostile feelings towards the hypnotist.

Q. Two final questions: First, will hypnosis replace the use of novocain?

A. This is very unlikely. The specific indications of each will certainly be further clarified in future. There may be considerable overlap in their indications. However, hypnosis greatly reduces tension and anxiety whereas novocain, while it may inhibit pain, frequently leaves the patient with considerable tension. Hypnosis should really be considered as one more element in the dentist's armamentarium.

Q. How to select patients for hypnosis?

A. Here again we need a great deal of study. Obviously in patients allergic to novocain and those who are exceedingly tense, anxious and frightened, hypnosis should be considered. Again, patients who are very disturbed or who have essentially neurotic problems must be dealt with judiciously and the dentist must not succumb to the temptation to attempt to alleviate their symptoms by direct suggestion in hypnosis.

CULTURAL COMPONENTS IN PAIN RESPONSE

Let us leave for a moment the essentially psychological influences on pain reaction to consider cultural components of response to pain. In this area we are particularly indebted to Dr. Mark Zborowski[2] who undertook research using standard anthropological techniques of interviewing and observation on veterans during the years 1951-56. The subjects of his study were members of different ethnic groups, first and second generation Jews, Italians and Irish, and "Old Americans"—that is, Anglo-Saxon white Protestants, whose families had been in America for three or four generations. The cultural importance of pain has been known for generations. Self-inflicted pain or self-mutilation in some countries has a well-defined purpose such as achieving special status in a society. In Germany, for instance, members of certain military student organizations would cut their faces with a razor in order to acquire scars that would identify them as members of a distinctive social group. We have discussed two different attitudes towards childbirth in our American culture. Generally, we know that in the United States, pain of childbirth is not welcome and various means are used to alleviate it. However, in Poland, pain of childbirth is not only expected, but accepted, and consequently little or nothing is done to relieve it.

In Zborowski's study, the majority of patients

[2] Zborowski, Mark. Cultural Components in Responses to Pain. Journal of Social Issues, Vol. VIII, No. 4, 16-30, 1952.

suffered pain from neurological diseases, mainly herniated discs and special lesions.

PAIN IN PATIENTS OF JEWISH AND ITALIAN ORIGINS

Members of both groups were described as being very emotional in their response to pain. They were described as exaggerating the pain experience, and being very sensitive to pain. Some doctors felt that Jews and Italians actually have a lower threshold of pain than members of other ethnic groups, especially those of Nordic extraction. (According to studies of Harold Wolff, however, threshold of pain is more or less the same regardless of nationality, sex or age.)

Underlying attitudes towards pain, however, are different in Italians as compared to Jews. Italian patients are mainly concerned with the immediacy of the pain experience; Jews focus mainly on the symptomatic meaning of pain, and on the significance of pain to their health and welfare and the welfare of their families. The Italian patient complains in relation to the pain as such; the Jewish patient expresses primarily his anxieties and worries as regards possible threats to his health.

Attitudes towards pain-relieving drugs were characteristic. When in pain, the Italian calls for relief; and his concern is with the analgesic effects of the drug administered. Once the pain is relieved he easily forgets his suffering and manifests a happy and joyful disposition. The Jewish patient, however, often is reluctant to accept the drug, and explains his reluctance in terms of concern about the effect of drug upon his health in general. He is apprehensive about the habit-forming aspects of the analgesic. Moreover, he feels that the drug relieves his pain temporarily and does not *cure* him of the disease. Thus many Jewish patients after being relieved may display the same depressed and worried behavior because no cure was effected.

Attitudes towards the doctor differ. The Italian displays a most confident attitude which is usually reinforced after the doctor has succeeded in relieving pain. The Jewish patient is skeptical concerning the doctor's ability to handle the basic illness so that he often tends to check the diagnosis and treatment of one doctor against another. The Italian, in a word, is present-oriented.

Both the Italian and Jew feel free to express emotions and feelings about pain—and this is consistent with their cultural values. They are not ashamed of this expression. They call for help and expect sympathy and assistance. When in pain they are reluctant to be alone, and prefer the presence and attention of other people. Americanized people tend to see this behavior as exaggerated and over-emotional, and the uninhibited display of reaction to pain provokes distrust instead of sympathy.

PAIN AMONG PATIENTS OF "OLD-AMERICAN" ORIGIN

There is little emphasis on emotional complaining about pain among "Old American" patients. They try to assume a detached role of accurate reporting and most efficient description for correct diagnosis and treatment. Withdrawal from society seems to be a frequent reaction to strong pain. Moaning and groaning are indulged in far less; the attitude being "It won't help anybody." When surrounded by members of family, friends, etc., they attempt to minimize pain and avoid complaining and provoking pity. The "Old American" tries to avoid being a nuisance. However, he, like the Jewish patient, is health-conscious and is prone to interpret his pain as a warning that something is wrong with his health. Pain is considered bad and unnecessary, and therefore must be immediately taken care of. A difference between the Jewish and "Old American" attitude, however, is this: the Jewish attitude is characterized by pessimism or skepticism in his future orientation; the "Old American" is optimistic in his future orientation, and there is faith in the progress of science. The "Old American" has a more positive attitude toward hospitalization because the hospital is the adequate institution equipped for necessary treatment. The Jewish and Italian patient may be disturbed by the impersonal character of the hospital and may prefer to be treated at home.

The patterns described above are most consistently manifested among immigrants. Their

descendents modify the original pattern of reaction in the direction of adopting the American form of behavior. The further the individual is from the immigrant generation, the more American is his behavior. A Jewish or Italian patient born in this country of American-born parents tends to behave like an "Old American" although he often harbors attitudes similar to those of the general Jewish or Italian culture.

The occupational factor plays an important role when pain affects a specific area of the body. For example, headaches cause more concern among intellectuals than among manual workers, whereas a herniated disc causes more disturbance for a manual worker than a professional or business person because of the relationship of the specific pain to ability to earn a living.

Education plays a role. The more educated patients are more health conscious and more aware of pain as a possible symptom of a dangerous disease.

Attitudes towards pain are acquired by individual members of the society from earliest childhood from values and concepts learned from parents and family members. Thus in Jewish and Italian families there are numerous reports of overprotective and overconcerned attitudes towards the child's health. These children are constantly reminded of the advisability of avoiding cold, injury, and fights or other threatening situations. Crying is responded to by parents with sympathy and help. By overprotection and worried concern, they foster complaint and tears. In the "Old American" family the parental attitude is quite different. The child cannot run to mother but is told to take pain like a man. The child is taught to expect to be hurt in sports and games and to fight back when attacked.

These differential learned adaptations to pain may lay the groundwork for stress and maladjustment for if the patient's attitude to pain conflicts with general cultural attitudes which surround him, the possibility of feelings of strain, self-consciousness, and not belonging are created; these feelings frequently lie behind emotional disturbance and neurotic adaptation.

PAIN IN THE NEUROPSYCHIATRIC DISORDERS

Thus far we have been concerned with variations of pain tolerance or reaction in essentially normal subjects, as the result of psychological or social influences. Now we come to the vast domain of the neuropsychiatric disorders. Pain may be suffered by patients with any type of clinical emotional disturbance from mild anxiety to the most severe schizophrenic disorganization. As with normal individuals, there may be exaggeration or distortion of pain based on lesion pathology, or there may be complaint of pain unrelated to any medical or neurological disorder. In clinical practice we never assume that pain is "an invention," but rather that it is a real part of the patient's problem; we rarely minimize the pain unless it appears specifically therapeutically indicated and justified.

Review of the literature in neuropsychiatric patients, I am sure, leaves the average dentist confused, as it does me, because of the great variety of pains suffered, the different clinical contexts in which pain occurs, and particularly the differences in psychodynamic mechanisms involved. Perhaps a little order can be introduced by conceiving of the patient's complaint of pain as being a special kind of communication, often unconscious, to the doctor, family or world at large.

The question is, What is the patient trying to communicate? We can often assume that the pain communicates very deep personality needs. For example, a patient, a young man of college age, who tells of pain in the chest region and shortness of breath, six months after a parent has died of acute coronary thrombosis, may be considered a case of perhaps mild anxiety neurosis with somatization. However, after a few psychotherapy sessions we find this young man is trying to communicate as follows: "I was brought up by an anxious mother who was always over-solicitous about my health. Her anxieties were communicated to me. Both mother and father had great respect for the heart as a potential organ of disease. We feared heart disease because there was a history in my father's family. I have been closely identified with my father and have unconsciously thought

of myself as following in his footsteps. Therefore, when father had several episodes of anginal pain, followed by fatal coronary, both unconsciously and consciously I have associated every kind of chest pain with impending disaster even though I intellectually appreciate that all my symptoms are minor and inconsequential."

YOUNG LADY FEARS CANCER

A young lady of more compulsive tendency but with considerable anxiety comes complaining of pain in her tongue and fear of cancer. As a child she felt the stern lash of a tyrannical Italian father. She was so rigidly reared as to make the expression of sexual or aggressive wishes not only taboo but liable to the most fearful punitive consequences. What is this lady communicating? Perhaps a reading of the unconscious mind would be as follows: "I have great trouble managing my sexual and aggressive wishes. I would like to express myself freely but I know that father would be enraged. Particularly in the sexual area, I am frightened at some of my fantasies or wishes involving oral pleasure. In a secret date with a boy-friend a year ago, I did things which bothered me and which I believe my parents would not countenance; yet the troubled events recur in my mind, and I can't stop thinking about them. It is less unpleasant to have oral pain than to face these thoughts. Anyway I get a lot of attention and solicitude which tends to reduce my guilt. After all, I'm not absolutely certain, am I, that I do not have cancer? I remember my Aunt Marie who had pain something like mine and later was discovered to have had a large thyroid for which surgical treatment was necessary."

Another example might be a very chronically ill lady of 42 who, following an argument with a house painter, fell prey to the most distressing "banging in the head." She was hospitalized in a panic state, given a course of electric shock with slight improvement, then underwent 2½ years of intensive psychotherapy by a competent psychiatrist. On several occasions her headache and agitation were so severe she again received EST. This lady recently underwent partial prefrontal lobotomy with some reduction of her anxiety and pain for three months followed by exacerbation.

Psychiatric exploration revealed that this lady always had a marginal adjustment. She is frightened of people, especially of her relatives; is very tense in their presence; feels that she is a tragic failure as a wife and a mother of two; she has been sexually cold throughout many years of marriage. To use her own words, "I am like a board," always thinking of unrelated things during sexual activity. This patient had a remarkably deprived early life, having lost mother and father at an early age and having been buffeted about from one unaccepting home to another. Extremely sensitive, she bore all the slights and rejections with fear and resentment unexpressed because of her extreme dependency. She is a woman full of anger and bitterness and deeply repressed but unsatisfied desires.

The persistent pounding headache and other somatic complaints are a communication to us of a feeling of disillusionment and failure, not only about lost opportunities but also regarding lost ability to enjoy herself. The fight with the house painter that marked the onset of acute symptoms mobilized overwhelming aggression which could not be handled and was converted into chronic hypochondriacal complaints and agitation.

These examples could be multiplied endlessly. Perhaps they give some insight into the great depth and meaning of pain—meaning that can be unravelled only through a prolonged doctor-patient relationship and careful and uncritical listening and understanding. Not only psychiatrists but specialists in medicine, dentistry, surgery and other human services have gradually learned the great power of the doctor-patient relationship if properly structured and the depth and complexity of emotions behind such apparently simple human communication as pain.

In helping our fellow man, beyond assets of technical skills and clinical experience we must add ourselves as potent therapeutic tools.

Harvard Medical School,
Boston, Mass.

DOLOR DE ORIGINE NON-ORGANIC

Dr. med. Milton Greenblatt

SUMMARIO IN INTERLINGUA

Ben que le phenomeno del dolor non es indispensabile pro le vita, illo ha un function importante in diriger nostre adaptation al influentias exogene e nostre sensation de ben-esser general como etiam in formar e determinar le structura de nostre personalitate. Le complexe problema del dolor e de su signification es possibilemente simplificate per considerar specificamente duo aspectos de illo: (a) le question del perception de dolor e (b) le question del reactiones evocate per illo.

Terminationes de dolor ha essite demonstrate in le pelle e in altere organos. Lor receptores fundamental es fin terminationes nervose medullate que se ramifica satis liberemente in areas sensibile. Iste terminationes nervose representa le componentes peripheric de elementos neural cuje componentes central passa via corno dorsal e tractos spino-thalamic al latere contrari verso nucleos thalamic, ab ubi neurones del proxime ordine continua primarimente verso cortice cerebral, specialmente in le area post-central. Le conscientia o cognoscentia de dolor ha su origine ab structuras supra le nivello dei ponte superior, del thalamo, del hypothalamo, e del cortice cerebral. Lesiones del ponte superior aboli permanentemente le appreciation de dolor como effecto de stimulation peripheric. Le thalamo es capace a appreciar dolor, sed apparentemente illo provide un ordine inferior de differentiation de dolor per combinar lo con altere modalitates. Ab studios de lesiones in humanos nos sape que le dolor thalamic es peculiar, distorte, e associate con disagradabile tonos de sensation. Al plus alte nivello cortical, sensationes de dolor es multo localisate in le areas del corpore e colorate per associate sensationes de tacto e pression. A iste nivello, le stato de excitation e de activitate del cerebro influentia le experientia de dolor.

THE INTRADERMAL ASCORBIC ACID TEST PART II. A REVIEW OF HUMAN STUDIES*

J. B. DUNBAR, D.M.D.†
E. CHERASKIN, M.D., D.M.D.**
and
F. H. FLYNN, B.S., M.T., A.S.C.P.†

INTRODUCTION

In an earlier report,[1] an attempt was made to review the literature on the use of the intradermal ascorbic acid test as revealed in animal studies. This paper will concern itself with an analysis of the literature dealing with human subjects.

HUMAN STUDIES

Rotter,[2, 3] having already established in his animal studies that the 2,6 dichlorophenolindophenol decolorization was a chemical reduction phenomenon, decided to verify this by injecting the dye into detached portions of human mucosa (perineal and vaginal). He found that the DCPIP* reagent disappeared in a time comparable to that in the living organism. The next study which he undertook was that of determining the connection between the velocity of reduction and body ascorbic acid. He placed six persons on a scorbutic regime for ten days. The urine level of ascorbic acid and the dye disappearance time were performed daily. The dye was injected into an area of the forearm free from hairs and veins. He observed that the urine level of vitamin C gradually declined while the intradermal time increased slowly. After ten days, oral and intravenous ascorbic acid was given to these subjects. The urinary ascorbic acid level increased and tended to become constant while the decolorization time decreased. He felt that the most astonishing finding was an 80 to 90 percent reduction in intradermal time two minutes after an intravenous injection of 600 mgm. of ascorbic acid. These findings, he believed, established the fact that the cutaneous reaction was the direct result of the ascorbic acid in the body. He recommended a N/400 concentration and 2 mm. wheal of DCPIP as a result of his experience with different concentrations and wheal sizes. Using this technique, he maintained that a disappearance time of 5 minutes or less spoke for saturation; 5 to 10 minutes should be considered normal; over 10 minutes indicated hypovitaminosis C. Rotter submitted that this procedure was better suited than urine or blood determinations for large population studies.

PORTNOY AND WILKINSON

Portnoy and Wilkinson,[4] in an experiment on 103 patients, employed Rotter's method of dye injection into the forearm. They used a N/400 concentration, injected .01 cc. to raise a 2 mm. wheal. To minimize technical errors, four wheals were raised in every instance and the average decolorization time established. From the entire group of 103 subjects, 35 were selected and then these were divided into two categories (21 unsaturated and 14 partially saturated) on the basis of plasma concentration. The unsaturated group (plasma levels of .27 to .52 mgm. percent) showed decolorization times of 3 to 29 minutes (mean 16.9). The partially saturated individuals (plasma levels of .72 to 1.3 mgm. percent) had times of 3 to 13 minutes (mean 7.5). Seventeen patients, taken from the unsaturated category, were then saturated (final plasma levels of 1.32 to 2 mgm. percent). The time range diminished to 0.5 to 6 minutes (mean 2.3). In every instance there was a decrease in intradermal time after

* From the Section on Oral Medicine University of Alabama School of Dentistry.
† Trainee in Epidemiology and Biometry, National Institute of Dental Research.
** Chairman, Division of Oral Surgery and Oral Medicine, University of Alabama School of Dentistry.

* 2,6 dichlorophenolindophenol

saturation. On the basis of a dietetic history, the remaining 68 subjects were categorized as saturated (18 persons), normal (37 persons), and unsaturated (13 persons). The mean intradermal times for these three categories were found to be 3.4, 8.8, and 16.1 minutes, respectively. This, it should be recalled, was consistent with the observations made on the original 35 subjects. The authors recognized the fact that there are individual variations. However, they concluded that the intradermal decolorization time is a useful and rapid indicator of the ascorbic acid status of the tissues. Consistent with Rotter's standard, these authors contended that an intradermal time of 5 minutes or less is indicative of tissue saturation; times over 10 minutes speak for deficiency.

Portnoy and Wilkinson,[5] in an enlarged study of 107 patients, investigated six different methods of vitamin C determination. These subjects were divided into the following groups: (1) two control categories of 51 patients, (2) one group with peptic ulcer (25 patients), and (3) peptic ulcer patients with hematemesis (31 subjects). It became clear that all measures of ascorbic acid status correlated with each other in an overall appraisal. In other words, those individuals with a presumably normal ascorbic acid body level showed higher plasma and urine levels and shorter intradermal decolorization times. Their evaluation of the intradermal time has already been considered (4). The results of this phase of their study may be summarized in six statements. First, the daily excretion of ascorbic acid proved to be greatest in the two control groups. Specifically, in staff personnel the average vitamin excretion amounted to 29 mgm. per 24 hours; in the other control group consisting of random ward patients, the mean daily urinary excretion was 17 mgm. In contrast, only 7 mgm. per day was excreted by the ulcer patients. Secondly, these investigators determined the saturation of their patients on the basis of a urine saturation test. According to the test, when the daily urinary excretion level reached 50 percent of an oral dose of 500 to 1000 mgm. of ascorbic acid, the subject was regarded as being saturated. On the basis of such a standard, Portnoy and Wilkinson found that the control groups required less supplementation with ascorbic acid to reach the 50 percent "spill-over" period than the experimental groups. Actually, this end point in the control groups was reached after a total of 500 to 2300 mgm. of the vitamin were administered. In contrast, 2100 to 8000 mgm. were required before one could demonstrate the 50 percent excretion in the experimental group. Thirdly, it was observed that the fasting plasma ascorbic acid levels in the control groups ranged from .60 to 1.85 mgm. percent whereas the peptic ulcer categories varied from .14 to .59 mgm. percent. Fourthly, observations were made using a 1000 mgm. intravenous load dose. It was noted that a maximal plasma level was reached 15 to 20 minutes after the intravenous dose. The 24-hour urinary vitamin excretion after a 1000 mgm. intravenous injection totalled 660 to 985 mgm. in the normal groups and 72 to 458 mgm. in the experimental subjects. Parenthetically, the statement can be made that about 80 percent of the total urinary excretion occurred in the first five post-injection hours. Fifthly, following a single oral dose of 1000 mgm. of ascorbic acid to a normal subject, the plasma level returned to its original value in 5 to 6 hours. In contradistinction, the return among the experimental subjects was found to occur in less time. Sixthly, Portnoy and Wilkinson observed that the decolorization time agreed quite well with the other laboratory tests though they preferred to regard the skin test time as only a rough estimate of saturation. The authors concluded with the statement that the evidence suggests that ulcer patients suffer with a severe ascorbic acid deficiency probably due in part to the ulcer diet which is customarily quite low in its ascorbic acid content.

PONCHER AND STUBENRAUCH

Poncher and Stubenrauch[6] performed intradermal times and plasma levels on 41 persons. Rotter's technique was employed. The subjects were arranged into three categories according to the plasma level of ascorbic acid. Group 1 (6 persons) varied in plasma concentration from .15 to .45 mgm. percent and were re-

garded as the scorbutic group. Group 2 (9 persons) showed a range from .5 to .7 and were considered subnormal. The normal group (26 persons) varied from .8 to 1.8 mgm. percent. No correlation was found between the dye decolorization time and ascorbic acid tissue saturation as evidenced by the plasma level. In the scorbutic group, the decolorization times were 4 to 8 minutes (average 5.8), in the suboptimal group 6.8 to 12 minutes (mean 9.4), and in the normal group 3.5 to 13.1 minutes (average 7.6). The authors stressed the fallacy of drawing conclusions from one test on one individual. Although they found no value with the procedure, Poncher and Stubenrauch reported that the decolorization times were constant for each individual.

JETTER

Jetter[7] found no correlation between the fasting ascorbic acid level of whole blood and the intradermal time in 50 female tuberculous patients. It is of interest that Jetter differed from his predecessors in that he used a N/500 DCPIP solution, .01 cc., and a 2 to 3 mm. wheal. Three wheals were raised on the forearm at the time the blood was drawn and the average of the three was reported. The intradermal times ranged from 2 to 10 minutes. The plasma values varied from .40 to 1.39 mgm. percent. Assuming 1.1 mgm. percent whole blood ascorbic acid as the minimal normal level, he divided his subjects into two groups: (1) 43 hypovitaminotic, and (2) 7 normal patients. The deficient patients evidenced skin times from 2 to 9 minutes (average 4.7) and the normal group decolorized the dye in 2 to 10 minutes (average 6.3). This investigator felt that vitamin C plays little or no role in the tissue decolorization of DCPIP.

CODVELLE ET. AL.

Codvelle *et al.*,[8] injecting intradermally .016 cc. of a N/200 (80 mgm. dye in 100 grams of diluent) DCPIP solution into the forearm of human subjects, found decolorization times between 5 and 22 minutes in 9 persons of the same age (20-23 years) and diet. In a second group of 5 normal patients given 100 mgm. of ascorbic acid daily for 15 days, the final disappearance times lay between 8 and 10 minutes. No initial times were given. A third group of 5 patients, who had received 500 mgm. of ascorbic acid for 6 days, ranged in time from 3 to 5 minutes. Again no initial values were given. Even though their method represented an extensive modification of Rotter's original technique, they designated the three groups as deficient, normal, and saturated respectively on the basis of Rotter's times. The authors then took groups comparable in age and with identical diets and performed urinary excretion tests of saturation at various levels of daily ascorbic acid intake. In the cases receiving a 100 mgm. supplement (9 persons) and in other groups receiving 150 or 200 mgm. (? persons), there was no abrupt increase in urinary excretion over a period of 30 days. A sudden increase of urinary excretion of the vitamin was held to indicate body saturation. Five persons receiving daily dosages of 250 mgm. showed a rapid rise in urinary excretion in from 4 to 7 days. Six subjects receiving 500 mgm. displayed the first massive excretion in 3 to 5 days. Assuming that this sudden rise of urinary ascorbic acid excretion denotes saturation, the group-nutrition of vitamin C would seem to be well related to the group-intradermal test time. Both the intradermal times and the urinary excretion would appear to speak for unsaturation in the non-supplemented and the 100 mgm. supplemented groups, whereas the 500 mgm. groups would appear to be saturated from either aspect. The authors were primarily interested in establishing an optimal daily ascorbic acid intake. They felt, on the basis of these and other findings, that 100 mgm. per day is needed.

GAMBIGLIANI-ZOCCOLI AND LOMBARDO

Gambigliani-Zoccoli and Lombardo[9] utilized the technique of Rotter in evaluating the intradermal test. The recorded decolorization time was an average of four wheals produced simultaneously on the forearm. In an unspecified number of healthy human subjects, 500 mgm. of ascorbic acid were introduced intravenously. The investigators observed that the time range,

before the injection of the vitamin, was 4 to 13 minutes. One hour later, the disappearance time decreased to as little as 2 to 3 minutes. The times returned to the normal range after 15-24 hours except in a few cases which required 48 hours to regain the original score.

OLIVEIRA LIMA

Oliveira Lima[10] employed Rotter's technique in estimating the value of the decolorization test. Twenty-four children (age 3-12 years) were separated into two groups (12 in each category), saturated and unsaturated. The saturated subjects excreted in the urine 40 percent of a 300 mgm. intravenous dose of vitamin C in three hours. The unsaturated group eliminated only 10 percent of the same dose in the same time. Prior to vitamin C injection the times ranged from 4 to 13 minutes (average 8) in the unsaturated group and from 6 to 11 minutes (average 9.7) in the saturated category. A scatter diagram revealed no relationship between the intradermal time and the urinary excretion pattern. The decolorization times were plotted against both absolute elimination values and percentage (of test dose) excretion within three hours. Oliveira Lima asserted that the test had no value. He suggested that the other reducing agents in the skin supervene and render the test, as offered, valueless.

POULSEN AND LIECK

Poulsen and Lieck[11] determined skin test times and ascorbic acid levels in 70 subjects. They used a N/300 (approximate) solution of DCPIP and a .01 cc. injection. They found times of 6 and 13 minutes in the same person when two wheals were raised simultaneously. This prompted them to standardize the injection at .01 cc. with a syringe modified to accomplish such exactitude. Then a double determination was done in 64 cases. None of the subjects showed differences in score greater than 2 minutes. The 70 experimental subjects, on whom plasma ascorbic acid levels and skin test times were performed, varied from 6 to 20 minutes in intradermal time and from 0 to .8 mgm. percent plasma level. The skin times were performed with the patients lying down and arms alongside the body. It was observed from a scatter diagram that decolorization times were not associated with plasma levels. For example, a decolorization time of 9 minutes corresponded to a plasma range of .08 to .8 mgm. percent. In another study, four subjects were supplemented with ascorbic acid for 11 to 42 days. The plasma ascorbic acid level rose from 0-.12 to .1-.8 mgm. percent. The skin times decreased from 8.5-15 to 8-12.5 minutes. The authors neglected to state how much ascorbic acid was administered, although they indicated that it was given orally. A load dose determination was done in one case. The fasting scores were .8 mgm. percent and 9 minutes. Two and three hours after the oral administration of 600 mgm. of vitamin C, the plasma levels were 1.15 and 1.6 mgm. percent respectively. On the other hand, the intradermal time decreased only slightly in 2½ hours—from 9 to 7 minutes. It is well to note that this patient had been taking supplementary vitamin C for 26 days before the load test. The authors pointed out that they failed to corroborate the finding of Rotter of an 80-90 percent reduction in time after a tolerance dose of ascorbic acid. To illustrate further the many variables which modify the skin test, Poulsen and Lieck injected both arms of one subject simultaneously and then had the patient raise one arm and lower the other. The higher arm decolorized the dye in 24 minutes; the other accomplished a decolorization in 16.5 minutes. Another patient was similarly injected and both arms were kept at the same level. However, one was immersed in a waterbath at 40° C. The intradermal time in the immersed limb was 14 minutes; the non-immersed arm, 18 minutes. An intradermal dye injection in the patellar skin of this same patient decolorized in 19 minutes. The authors concluded, from these observations, that the skin test time has no clinical value.

WRIGHT AND MACLENATHEN

Wright and MacLenathen[12] studied the intradermal time from two aspects: (1) the correlation of diet and blood level, and (2) relationship of site of injection to disappearance

time. Of 11 patients with adequate diets and normal blood levels, only three had decolorization times of 10 minutes or less. According to Rotter,[2, 3] individuals with adequate blood levels should have shown disappearance times between 5 and 10 minutes. Of 11 patients with poor dietary habits and subnormal blood levels, 4 showed intradermal times less than 10 minutes. Of the latter group of 11, the remaining 7 subjects displayed disappearance times ranging from 11 to 33 minutes. The authors did not state which blood level was considered normal nor did they list the observed plasma levels. It should be emphasized that the research of these men is at variance with Rotter[2,3] even though the same dye, in the same concentration, was used. Wright and MacLenathen also compared decolorization times at different sites of injection. In some of the subjects utilized in the preceding experiment and in a group of individuals with vascular disease there was a difference in the test times when two wheals were simultaneously raised within a few centimeters of each other. For example, in one instance the disagreement between the decolorization times of concurrent dye injections amounted to ten minutes. Parenthetically, the authors pointed out that intense pain occurs at the site of injection and that there remains a hard, erythematous nodule for 4 to 5 weeks.

BANERJEE AND GUHA

Banerjee and Guha[13] studied 9 human beings using Rotter's original technique. Each subject was given an oral dose of 700 mgm. ascorbic acid. Intradermal times and 24-hour urine ascorbic acid excretion values were performed before and after administration of the load dose. Eight of the 9 subjects showed a decrease in test time and all displayed increased urinary excretion. In these 8 cases, an average reduction of 35 percent of the original skin time resulted. It is interesting to note from their chart that the longer original times showed the greatest average percentage reduction.

SUZUKI

Suzuki,[14] using Rotter's method of intradermal injection[2,3], compared blood serum values with the decolorization times. He employed eight groups of a mixture of healthy persons and individuals afflicted with skin diseases. He did not, however, reveal the number of persons in each group. The groups were divided into increasing levels of vitamin C in the blood serum. He found a direct relationship between the two scores—the intradermal times became lower just as the serum level increased. He noted in seven cases of lichenifying and pigmenting skin diseases that the dye disappeared in a shorter time when it was injected into an area of the skin involved by the disease. Times were longer when the dye was injected into an uninvolved skin site. In eight patients with urticarial wheals he injected the dye into unaffected areas, into the halo area between the wheal and the surrounding skin, and directly into the wheal itself. Proceeding from the normal to the involved area in this manner, a progressive decrease in test time occurred. He felt that these observations could be explained on the basis of a local increase in "reducing substance" due to acute or chronic inflammation.

GOLDSMITH, GOWE AND OGAARD

Goldsmith, Gowe and Ogaard[15] made 100 observations on 45 persons. These individuals were classed into three groups: (1) unsaturated, (2) saturated, and (3) normal. The classification was based on the level of ascorbic acid in the plasma. Using .01 cc. of a N/400 solution of DCPIP, they claimed no correlation between the plasma ascorbic acid level and the intradermal time. They, therefore, concluded that the intradermal test cannot replace blood determinations as a measure of vitamin C status. They found that factors other than the ascorbic acid level could modify the decolorization time. This observation was derived from studies in which they instituted changes in circulation and room temperature. The authors further felt that other reducing agents might interfere with the disappearance time of DCPIP. An analysis of data from 9 persons who had received more than 5 injections was made. Once again, the authors found no correlation between the test time and plasma level. For example, in one subject given 25 intradermal injections, within

75 minutes, there was a time range from 4.5 to 11.6 minutes. On the other hand, they observed that the intravenous administration of 1000 mgm. of ascorbic acid caused a plasma level rise from .15-1.4 to 6-8 mgm. percent. During this same interval the intradermal time was halved. They failed to indicate the number of cases anlayzed and the time interval between injection of the load dose and the final measurements.

SLAVICH AND TORRINI

Slavich and Torrini[16] performed Rotter's test on 50 individuals. They employed 36 human beings in a loading study. The group contained normal and sick persons. The intradermal time of each person was taken, and then varying amounts of ascorbic acid (200-250-500 mgm.) were given intravenously. Fifteen to 20 minutes later, the skin test time was repeated. The subjects fell into three groups on the basis of original times: (1) 14 revealed skin times below 10 minutes (range 3.5-10 minutes), (2) 9 had times between 10 and 14 minutes (range 11-13 minutes), and (3) 13 exhibited decolorization times above 14 minutes (range 14-35 minutes). In group 1, 4 persons were apparently in normal health and 10 were diseased. Group 2 was composed of 4 normal and 5 sick patients. In group 3 all were ill. It is interesting to note that the decolorization times found after ascorbic acid administration were lower in every case than the preinjection scores. And further, the average percentage reduction in time was greatest in the groups which originally showed the longest disappearance times. Specifically, group 1 revealed an average reduction in skin test time of 42 percent, group 2 an average of 46 percent, and group 3 (with the longest original times) presented an average of 50 percent reduction. The authors felt that the other reducing agents present in the skin did not lessen the clinical value of the intradermal test. They believed that the time obtained reflects body saturation since the integument acts as a storehouse for the vitamin. The other reducing agents (as glutathione and cysteine) seem to remain constant within any individual even though the ascorbic acid may fluctuate. Slavich and Torrini, however, did agree that each individual should be viewed singly. These authors implied that load testing with calculation of percentage reduction of time may be an indication of body ascorbic acid status.

BARALDI

Baraldi[17] employed Rotter's method in performing the intradermal test on 84 mentally-ill patients, ranging from 15 to 85 years of age. He obtained times between 5 and 8 minutes in 80 of the 84 patients. Two subjects revealed times below 3 minutes and two above 13 minutes. The author noted that these times were well within the physiologic limits set by Rotter, except for the two cases above 13 minutes. According to the author, the skin test time did not distinguish between the new admissions to the institution (22 persons) and those who had been there some time. The new admissions were alcoholics and other malnourished persons. The old patients had, Baraldi submitted, enjoyed a nourishing diet. For this reason, the investigator contested the specificity of the intradermal test.

BECK AND KRIEGER

Beck and Krieger[18] utilized Rotter's method in evaluating the skin time in human beings. In preliminary studies they found that variations in both blood supply to the tissue and in light intensity had no appreciable effect on the disappearance time. They performed the decolorization time before and after cutting off blood circulation in the arm (pressure cuff), before and after intensive rubbing of the skin and before and after cardiazol injection (which decreases peripheral blood circulation). Under these circumstances the time-scores differed in value by only 1 to 2 minutes. Upon injecting the dye different depths into the skin on the same person simultaneously, they observed differences in time of over 50 percent. This led them to fabricate an apparatus whereby a constant depth of puncture could be achieved. The intradermal time was then determined in 62 presumably healthy persons. They ranged from 5 to 16 minutes with an average value of 10 minutes. Three persons who were experimental-

ly saturated with vitamin C (urine level verification) showed diminished times (4 to 5 minutes). In 16 subjects from the first study a load-dose experiment was done. The subjects were given 300 mgm. of ascorbic acid per day, per os, until the urine concentration of the vitamin reached a particular level. They indicated that this level in the urine was normally attained after 5 days — i.e., after a total dose of 1500 mgm. The amount of ascorbic acid required to reach the desired urine concentration was then compared with the initial disappearance time. There was a close relationship—persons with longer original times required greater amounts before urine saturation was attained. It is interesting to note from their graph that the usual amount of vitamin C necessary to saturate the tissues (1500 mgm.) is related to an initial disappearance time of 12 minutes. In a larger study of 92 patients, Beck and Krieger distinguished two groups: (a) 52 persons who had illnesses known to be unaccompanied by ascorbic acid deficiency, and (b) 40 subjects who had diseases characterized by ascorbic acid deficiency. The average disappearance time was found to be 10.5 and 18.5 minutes respectively. The tendency for group b to exhibit prolonged times was clear. In addition the authors performed skin times on 8 mothers and their newborn soon after delivery. The greatest difference in time between mother and child was 3 minutes. Beck and Krieger felt the Rotter test was valuable as a rough clinical estimate of tissue ascorbic acid.

RAPAPORT AND MILLER

Rapaport and Miller[19] performed plasma ascorbic acid levels and intradermal times on 100 children selected at random from the hospital wards. Using a N/400 solution of DCPIP, they raised 1 mm. wheals on the extensor surface of the thigh about 6 inches above the knee. These children were arranged into three groups according to their plasma ascorbic acid levels: (1) *low*—level below .5 mgm. percent (26 children), (2) *subnormal*—level .5 to .7 mgm. percent (21 children), and (3) *normal*—level above .7 mgm. percent (53 children). Group 1 (low) varied in intradermal time from 2.5 to 30 minutes (average 6.8 minutes). Group 2 (subnormal) showed an intradermal time range from 2 to 20 minutes (average 5.5 minutes). Group 3 (normal) varied from 2 to 20 minutes (average 5.4 minutes). Only in the normal range did they observe that the intradermal time allowed a fair estimation of the plasma level. In the other two groups, no predictive correlation was possible. For this reason, Rapaport and Miller deemed the disappearance test valueless for clinical purposes.

BAKHSH ET. AL.

Bakhsh et al.,[20] using Rotter's technique, performed plasma ascorbic acid levels and intradermal disappearance times in students. One hundred and forty determinations were done. Twenty cases (14.3 percent) fell below .8 mgm. percent in plasma value; 81 cases (57 percent) lay between .8 and 1.5 mgm. percent; 39 cases (27.9 percent) yielded plasma levels in excess of 1.5 mgm. percent. The times varied from 1 to 25 minutes and the plasma values from .3 to 2.0 mgm. percent. The mean standard deviations for the students were: Plasma level 1.19 ± .3 mgm. percent; skin time 8.2 ± 4.2 minutes. The authors established statistically that the two values were not independent of each other. However, they were distributed over such a wide area as to preclude any mutual predictivity. For example, a blood level of .8 mgm. percent corresponded to times from 7 to 19 minutes. In a second study the authors described the results of 200 determinations done on in-patients. The average plasma value was .88 ± .20 mgm. percent while the average intradermal time was 9.1 ± 3.6 minutes. Sixty cases (30 percent) had plasma levels below .8 mgm. percent; 138 cases (69 percent) revealed levels between .8 and 1.5 mgm. percent; only 2 cases (1 percent) had values above 1.5 mgm. percent. The times varied from 3 to 24 minutes and the plasma values from .4 to 1.6 mgm. percent. The two variables were found to be in a relation which could have arisen by chance. The coefficient of correlation for the student data was -.6522 and for the in-patients -.6111.

Of 61 "pyorrhea cases," no low plasma levels were noted. Bakhsh et al. concluded that the intradermal time cannot replace the plasma level for estimating vitamin C status of the tissues. Additionally they noted that the intradermal time for any one individual did not vary significantly.

BANERJEE AND GUHA

Banerjee and Guha[21] sought a minimal decolorization time in human subjects. Six healthy adult students were subjected to the skin test (Rotter technique) and then given an oral dose of 700 mgm. of ascorbic acid. On the following four days the intradermal time and three-hour urinary ascorbic acid excretion were measured. The initial time range of the students was 1 to 2.5 minutes (average 2 minutes). On the fifth day a minimum skin time of 90 seconds was found in every case but one, whose score was 85 seconds. According to the urinary saturation test, all students were saturated on the third day. It should be mentioned that this urinary saturation test claims body saturation where the urinary disposal of ascorbic acid reaches a peak. Banerjee and Guha felt, on the basis of these studies, that the skin test is a more valuable indication of body saturation with ascorbic acid than the urinary excretion method. This they based on the fact that the renal threshold may vary and that the diuretic action of vitamin C may promote its own excretion in large amounts before the body has time to assimilate it.

GLATT

Glatt[22] determined the plasma ascorbic acid values and intracutaneous decolorization times on 30 ill and convalescent infants ranging in age from 3 weeks to 5 months. Rotter's technique was used except that the thigh was the injection site. In 20 of the infants, he found plasma levels from .36 to 1.44 mgm. percent (which he regarded as normal) and intradermal test times ranged from 9 to 46 minutes. The remaining 10 cases were given small (amount not specified) ascorbic acid supplements in the diet. He found no significant differences in skin test times in this group compared with the other 20 subjects, although the plasma levels were higher.

ROSSINI AND FERRARI

Rossini and Ferrari[23] employed Rotter's technique in evaluating the intradermal test. Forty-seven pulmonary tuberculosis patients were studied from the standpoint of urinary saturation, capillary fragility, and intradermal time. They called attention to the fact that tubercular patients are recognized as deficient in vitamin C by all authorities. They stated that the deficiency status of the 47 patients was confirmed by the urinary saturation test and by the capillary fragility index. The urine saturation test consisted in determining the amount of ascorbic acid excreted within three hours after a 300 mgm. dose (route?) of ascorbic acid. A 40 percent excretion was accepted as normal; a 10 percent or less elimination was considered to be evidence of a deficiency state. They then proceeded to perform an intradermal time tolerance study. The decolorization time was determined for each patient. This was followed by an intravenous injection of 300-500 mgm. of ascorbic acid. One hour later, the test-time was again performed. The average disappearance time of four wheals raised simultaneously was taken as the true score. Initially the time-score range was 9-14 minutes. The postinjection values were from 6 to 9 minutes. The authors found that, within this group, the hemoptysis patients presented postinjection times longer than those who did not bleed. They concluded that a single skin test time is not informative.

UZAN

Uzan[24] stated that he had been able to confirm the parallelism between the dye decolorization time and the body ascorbic acid status. Although no description of method nor results was given, he avowed that the correspondence between the skin time and the urine saturation test was experimentally verified by him. He also agreed with Rotter that local changes in vascular flow (dilatation, constriction) or changes in temperature did not appreciably alter the decolorization time. He felt that the action of other reducing agents in the skin was relatively

weak. Uzan suggested that the skin test is an invaluable tool when other more complex methods are not available.

UZAN AND BRONSTEIN

In an abstract of an article by Uzan and Bronstein,[25] based on Rotter's technique, it was pointed out that times of 10 minutes or more denote deficiency; disappearance times of 2-3 minutes indicate health relative to vitamin C.

Uzan and Bronstein,[26] in another publication, simply confirmed their earlier observations.[25]

REDDY AND SASTRY

Reddy and Sastry,[27] in pilot studies on themselves, found that the decolorization time decreased from 10 minutes to 3 minutes 10 seconds, and from 6 minutes 40 seconds to 3 minutes 20 seconds respectively, after 5 and 7 days on a supplement of 70 mgm. ascorbic acid per stone weight (14 lbs.). They also noted that the intradermal time continued to diminish even after saturation was reached (on the basis of urinary excretion). These researchers used the Rotter method already described. In a series of medical students and young elementary school children, classified into three groups according to frequency of citrus fruit consumption, they found that those with the highest intake exhibited the lowest intradermal times, the scores being less than 10 minutes. Out of the 65 apparently healthy medical students, 48 (74 percent) had decolorization times between 5 and 9 minutes. Reddy and Sastry, consequently, agreed with Rotter that a 5 to 10 minute disappearance time signifies a satisfactory state of ascorbic acid nutrition.

Reddy and Sastry[28] performed the intradermal test on 69 ophthalmic patients, employing a N/400 solution and 2 mm. wheal. At the same time, they collected information on the dietetic habits of the subjects. These authors were not evaluating the intradermal test—they were utilizing it as a measure of vitamin C status of ophthalmic cases. They concluded that patients with inflammatory eye diseases (retinitis, corneal ulcer, trachoma, optic neuritis) are more frequently associated with a vitamin C deficiency than those with cataracts alone.

DOLLÉ

Dollé[29] measured the intradermal times (Rotter's method) of four healthy persons using as the variable dye solutions of different ages. In other words, it was possible to determine the disappearance time employing a freshly prepared solution and one which had been allowed to stand for one day. Under these conditions, he found variations of 3-4 minutes in decolorization times in each person. Dollé believed that this is due to a strong light sensitivity of the dye. It then follows that the postpreparation time of the test solution must be standardized. Dollé then performed intradermal times on each of 82 obstetric and gynecologic patients. No specific times are listed. However, the group with unusual surgical blood loss revealed the greatest number of subjects with prolonged disappearance times (values above 10 minutes). The converse was true in the normal group. The author then related these skin times to the clinical history of each patient regarding possible indications of a vitamin C deficiency (Rumpel-Leede phenomenon, gingival bleeding, general malaise). In about one-half of the subjects, there was a significant parallelism between the intradermal time and the clinical observation. Twenty-eight patients with prolonged times were given an unspecified amount of vitamin C per os. After one day, 6 showed normal times; after 6 days 14 revealed normal scores. Six other patients who had had initial decolorization times of 20 minutes decolorized the dye in 14 minutes at the end of the 6 day period. Six control persons not receiving the supplement showed consistently the same times throughout this study. Forty-three surgical patients were tested intradermally before and after an operation. The 6 patients with only minor surgical treatment showed no differences in pre- and postsurgical time. Of 37 who underwent extensive surgery (laparotomy), 16 showed increased postoperative skin times. Six others could not be evaluated since they had been injected intravenously with Ce-Ferro (an iron-ascorbic acid compound). In the remaining 15, no change was noted. Dollé concluded that the prolonged dye disappearance time is due to blood loss during

the operation, lack of absorption and poor appetite following surgery. The author felt that his results qualify the skin test as a rough index of ascorbic acid nutrition.

SLOBODY

Slobody[30] performed intradermal tests using varying wheal sizes and dye concentrations. He determined early that the 4 mm. wheal yielded the most constant test time when intradermal injections were performed simultaneously on the same forearm. Employing the 4 mm. wheal, he then introduced dye concentrations from N/1500 to N/300 intracutaneously in 167 patients. It became clear that the greater the concentration of DCPIP, the more reliable the test results would be. Not only would longer times lend themselves to easier interpretation, but the effects of other reducing substances present would be minimized. He subsequently employed a 4 mm. wheal (about .05 cc.) and a N/300 dye solution.* He performed 285 skin test times in 167 hospital patients in duplicate on the same and opposite forearms. At the same time, a blood vitamin C determination was done. The blood levels varied from 0 to 1.97 mgm. percent. The decolorization times ranged from 3.5 to 25.6 minutes. Fifty-nine blood levels were below .3 mgm. percent. In this group, 5 subjects exhibited times between 9.5 and 13.2 minutes; the remaining 54 showed times above 14 minutes. The mean skin test time for this category was 16.7 minutes. Since a correlation existed in only about 50 percent of the total number of patients, the author questioned the validity of plasma level as a determinant of nutritional status. The statement was made that the continuous fluctuating plasma ascorbic acid level reflected only recent dietary intake. In some patients, for example, who had taken vitamin C the previous day, high plasma levels were manifest while the intradermal time remained elevated. Forty-five cases were given 200 mgm. ascorbic acid intravenously following plasma and intradermal determinations. Thirty minutes postinjection the same measurements were repeated. In 44 of the 45 cases, the skin test time diminished and the plasma level of ascorbic acid increased. In addition, 10 children (aged 3 to 11 years) were placed on diets free of fruits and fruit juices. Plasma scores and intradermal times were determined weekly. The blood vitamin C level decreased with simultaneous increase in disappearance time. After 9 weeks, the skin test time exceeded 14 minutes. Biomicroscopic gingival examinations revealed changes attributable to hypovitaminosis C as described by Kruse[31]. Two of the children were subsequently given ascorbic acid and the skin test times returned to normal. The DCPIP time in one typical child, on this regime for 9 weeks, increased from 7.5 to 16.5 minutes. Within this same period, the blood level of ascorbic acid declined from 1.33 to .20 mgm. percent. Slobody suggested, on the basis of his recommended technique, with N/300, 4 mm. wheal and .05 cc., that a disappearance time above 14 minutes be regarded as evidence of pronounced unsaturation; 9 to 13 minutes as mild unsaturation; less than 9 minutes as proof of a satisfactory vitamin C status. He further suggested that the intradermal decolorization time might well be developed into a saturation index. This would be, he believed, a welcome and simple tool. It is of interest that some of his subjects reported a burning sensation during injection. Two of the 10 children developed nodules at the injection site which persisted for two weeks.

BANERJEE

Banerjee[32] found statistically insignificant variations in decolorization time in 6 individuals when the dye solution was injected into the same part of the arm on successive days, into adjacent sites on the same day, and simultaneously in opposite arms. Six subjects were given 200 mgm. of ascorbic acid daily for 3 weeks. The intradermal times diminished gradually from 5-12.5 minutes to 2.7-5.0 minutes. On the basis of his previous study,[18] in which he established that 90 seconds indicates saturation, he concluded that 200 mgm. ascorbic acid per day is not sufficient to produce saturation. He indi-

* A N/300 DCPIP solution is prepared by dissolving 24 mgm. sodium 2,6-dichlorobenzenoneinindophenol (Fisher) in 35 cc. of distilled water heated to 95° C. This solution, when cooled, is then diluted to 50 cc. with distilled water in a volumetric flash.

cated in this report that the skin test time varied with the season and with the availability of fresh citrus fruits and vegetables. It was Banerjee's opinion that the decolorization time is a fair index of group vitamin C status but only a rough estimate of the individual's ascorbic acid tissue level.

GOUNELLE AND VALLETTE

Gounelle and Vallette,[33] using a N/400 dye concentration and 2 mm. wheals, studied the whole blood ascorbic acid levels and skin test times in 88 human beings. The subjects included 38 controls, 41 apyretic and 9 pyretic tuberculous individuals. The whole blood ascorbic acid levels and disappearance times in the control group ranged from 0 to 17 mgm. percent and 2 to 12 minutes respectively. In the apyretic tuberculous group the scores were 0 to 13 mgm. percent and 2.5 to 13 minutes. The pyretic group exhibited values of 2.5 to 14 mgm. percent and 3 to 6 minutes. The investigators contended that there is no correlation between blood and skin test results and that the blood determinations remain the best index of latent hypovitaminosis C.

MOENA AND NOLFF

Moena and Nolff[34] employed the method of Slobody.[30] They modified the method by utilizing an 8.5 percent solution of sodium chloride as the dye solvent in place of distilled water. This substitution was found to reduce the burning pain associated with the injection. In the majority of cases, an indurated postinjection nodule persisted for 3 to 4 days. An interesting observation was that a characteristic red, pruritic papule appeared 10 days following the skin test and persisted 6 to 8 days. They felt this was an anaphylactic phenomenon. They performed the skin test in 1,150 persons. Of these, 845 were considered to be healthy individuals. The group was analyzed as a whole and in the light of such factors as age, sex, type of work. The authors evidently accepted the contention of Slobody[30] that disappearance times above 14 minutes indicate a subnormal ascorbic acid status. The findings in the 845 healthy persons disclosed 77 percent with skin time below 10 minutes, 21 percent between 17-20 minutes, and 2 percent 21-25 minutes. The authors concluded that, with advancing age, the intradermal time lengthened. No sex differences could be observed. They next undertook a study of 305 pathologic cases. It appeared to these investigators, from a study of intradermal time in patients with different disorders, that certain conditions (e.g. osteomyelitis and pregnancy) were not associated with an ascorbic acid deficiency. In contrast, in other pathologic states (e.g. typhoid fever, deficiency states, narcosis, chronic gastritis) 100 percent of the subjects showed significantly prolonged intradermal times. Thus, of the total 305 pathologic cases, 40 percent exhibited times below 14 minutes whereas 57 percent of the so-called healthy persons showed the same disappearance values. The authors then selected 162 patients on the basis of factors presumed to be associated with vitamin C deficiency. The evidence, from intradermal testing, disclosed that 60 to 90 percent of such individuals displayed decolorization times greater than 14 minutes. Another study was performed with 28 patients. They were given vitamin C orally or intravenously to observe the effect upon the skin time. No information as to dosage or time intervals was given. Specific figures were available in some cases but not in others. Fourteen persons who had original times over 14 minutes, declined to a level below 14 minutes after load dosing. Seven persons who were originally above 14 minutes, exhibited a 16 percent mean decrease (from an average of 19.7 minutes to 16.4 minutes). This group did not, then, fall below 14 minutes after the tolerance dose. Two persons who had received 500 mgm. of ascorbic acid for 2 and 3 days respectively, showed no change from the original score of 16 minutes. Five persons who were originally below 14 minutes declined in time an average of 19.5 percent (from a mean of 11.2 minutes to 9 minutes). All the patients except two showed a decrease in time. Another study was done to observe the changes in plasma ascorbic acid following a load dose. Oral administration elicited a rise in ascorbic acid blood level in one hour; the intramuscular route caused an in-

TABLE 1

Author(s)	*Amount of Injected Dye (cc.)*	*Concentration of Dye*	*Wheal Size (mm.)*	*Size of Injection*
Rotter (2, 3)	.01	N/400	2	forearm
Portnoy & Wilkinson (4, 5)	.01	N/400	2	forearm
Poncher & Stubenrauch (6)	.01	N/400	2	forearm
Jetter (7)	.01	N/500	2-3	forearm
Codvelle et al. (8)	.016	N/200	?	forearm
Gambigliani-Zoccoli & Lombardo (9)	.01	N/400	2	forearm
Oliveira Lima (10)	.01	N/400	2	forearm
Poulsen & Lieck (11)	.01	N/300	2	forearm
Wright & MacLenathen (12)	.01	N/400	2	forearm
Banerjee & Guha (13)	.01	N/400	2	forearm
Suzuki (14)	.01	N/400	2	forearm
Goldsmith et al. (15)	.01	N/400	2	forearm
Slavich & Torrini (16)	.01	N/400	1.5-2	forearm
Baraldi (17)	.01	N/400	2	forearm
Beck & Krieger (18)	.01	N/400	2	forearm
Rapaport & Miller (19)	?	N/400	1	thigh
Bakhsh et al. (20)	.01	N/400	2	forearm
Banerjee & Guha (21)	.01	N/400	2	forearm
Glatt (22)	?	N/400	2	thigh
Rossini & Ferrari (23)	.01	N/400	2	forearm
Uzan (24)	.01	N/400	2	forearm
Uzan & Bronstein (25, 26)	.01	N/400	2	forearm
Reddy & Sastry (27, 28)	?	N/400	2	forearm
Dollé (29)	.01	N/400	2	forearm
Slobody (30)	.05	N/300	4	forearm
Banerjee (32)	.01	N/400	2	forearm
Gounelle & Vallette (33)	?	N/400	2	forearm
Moena & Nolff (34)	.05	N/300	4	forearm
Zanotti (36)	.01	N/400	2	forearm
Viñas-Espin (37)	.025	approx N/300	?	forearm

crease in 30 minutes; the intravenous injection prompted an immediate climb. The ascorbic acid levels began to decline after about 4 hours in the case of intramuscular and oral routes, whereas the intravenous route was associated with an intense level for 2 hours, after which there was a decline. The authors measured the plasma ascorbic acid level twice in the same day on 16 patients following oral vitamin C. They stated that each individual exhibited the same pattern on both occasions. On the basis of plasma ascorbic acid determinations of 10 presumably healthy individuals, they established a plasma level of .8 mgm. percent or more as an index of health. Using this plasma norm and 14 or less minutes as a normal intradermal time, they studied 39 subjects. Twenty of the patients, with intradermal times above 14 minutes, showed plasma levels less than .8 mgm. percent and 11 subjects, with intradermal times greater than 14 minutes, displayed plasma levels above .8 mgm. percent. In contrast, only 1 of the 8 persons with an intradermal time below 14 minutes showed a plasma level below

TABLE 2

Author(s)	*Number and Health of Subjects*	*Duration of Experiment (Days)*
Rotter (2, 3)	6 (U)*	10
Portnoy & Wilkinson (4,5)	21 (U)	?
	14 (P)*	—
	18 (Sat)*	—
	37 (N)*	—
	13 (U)	—
Poncher & Stubenrauch (6)	26 (N)	—
	9 (U)	—
	6 (S)*	—
Jetter (7)	43 (U)	—
	7 (N)	—
Codvelle et al. (8)	9 (N)	—
	5 (N)	15
	5 (N)	6
Gambigliani-Zoccoli & Lombardo (9)	? (N)	1 hour
Oliveira Lima (10)	12 (Sat)	—
	12 (U)	—
Poulsen & Lieck (11)	70 (?)	—
	4 (?)	11-42
	1 (?)	2.5 hours
Wright & MacLenathen (12)	11 (N)	—
	11 (U)	—
	20 (U, N)	—
Banerjee & Guha (13)	9 (N)	1
Suzuki (14)	? (U)	—
	? (U)	—
	? (U)	—
	? (U)	—
	? (U)	—
	? (U)	—
	? (P)	—
	? (N)	—
Goldsmith et al. (15)	42 (N)	—
	26 (U)	—
	32 (Sat)	—
Slavich & Torrini (16)	14 (N, U)	20 min.
	9 (N, U)	20 min.
	13 (U)	20 min.
Baraldi (17)	80 (?)	—
	2 (?)	—
	2 (?)	—
Beck & Krieger (18)	62 (N)	?
	16 (N, U)	2-11
	52 (N)	—
	40 (U)	—
Rapaport & Miller (19)	53 (N)	—
	21 (P)	—
	26 (U)	—
Bakhsh et al. (20)	140 (?)	—
	200 (?)	—
Banerjee & Guha (21)	6 (N)	6
Glatt (22)	20 (N)	—
	10 (Sat)	7-14
Rossini & Ferrari (23)	47 (P)	1 hour
Uzan (24)	5 (?)	?
Uzan & Bronstein (25, 26)	? (?)	?
Reddy & Sastry (27)	21 (Sat)	—
	24 (N)	—
	20 (U)	—
Reddy & Sastry (28)	30 (N)	—
	20 (N)	—
	19 (N)	—
Dollé (29)	30 (U)	60
	42 (U)	60
	10 (N)	—
	5 (?)	?
	1 (?)	?
	24 (?)	?
	4 (?)	?
	6 (?)	?
Slobody (30)	167 (?)	—
	10 (N)	63
Banerjee (32)	6 (N)	21
Gounelle & Vallette (33)	38 (N)	—
	41 (U)	—
	9 (U)	—
Moena & Nolff (34)	845 (N)	—
	305 (P)	—
	14 (?)	?
	7 (?)	?
	5 (?)	?
	2 (?)	2-3
Zanotti (36)	50 (U)	—
Viñas-Espin (37)	? (?)	?

* U = unsaturated; P = partially saturated; Sat = saturated; N = normal; S = scorbutic

TABLE 3

Author(s)	*Initial Intradermal Time (Min.)*	*Final Intradermal Time (Min.)*	*Mode of Vitamin Administration*
Rotter (2, 3)	?	80-90% decrease	600 mgm. IV
Portnoy & Wilkinson (4, 5)	3-29 (16.9)	.5-6(2.3)	500-1000 mgm. P.O. q.d.
	—	3-13(7.5)	—
	—	2-7(3.4)	—
	—	3-16(8.7)	—
	—	4-33(16.1)	—
Poncher & Stubenrauch (6)	—	3.5-13.1(7.6)	—
	—	6.8-12(9.4)	—
	—	4-8(5.8)	—
Jetter (7)	—	2-9(4.7)	—
	—	2-10(6.3)	—
Codvelle et al. (8)	—	5-22	—
	?	8-10	100 mgm. daily, oral route?
	?	3-5	500 mgm. daily, oral route?
Gambigliani-Zoccoli & Lombardo (9)	4-13	as low as 2-3	500 mgm. IV
Oliveira Lima (10)	—	6-11(9.7)	—
		4-13(9)	—
Poulsen & Lieck (11)	—	6-20	—
	8.5-15	8-12.5	oral amount?
	9	7	600 mgm. oral
Wright & MacLenathen (12)	—	under 10 to 19	—
	—	under 10 to 33	—
	—	?	—
Banerjee & Guha (13)	1.2-8.2(3.8)	1.3-3.5(2.8)	one oral dose of 700 mgm.
Suzuki (14)	—	23.5	—
	—	17.8	—
	—	12.6	—
	—	9.5	—
	—	8.6	—
	—	8.0	—
	—	6.1	—
	—	4.4	—
Goldsmith et al. (15)	—	2-17.5(6.9)	
	—	2-14.7(8.7)	
	—	3-15.4(8.3)	
Slavich & Torrini (16)	3.5-10(9.6)	3-8(5.5)	200-250 mgm. IV
	11-13(11.8)	4-9.5(6.4)	200-550 mgm. IV
	14-35(21.8)	6-19(10.9)	200-550 mgm. IV
Baraldi (17)	—	5-8	—
	—	below 3	—
	—	above 13	—
Beck & Krieger (18)	5-16(10)	4-5	all were "saturated" with ascorbic acid
	4-20	?	300 mgm./day P.O. until saturated (urine test)
	—	19.3% above 12 min.	—
	—	92.5% above 12 min.	—

TABLE 3 (Continued)

Author(s)	*Initial Intradermal Time (Min.)*	*Final Intradermal Time (Min.)*	*Mode of Vitamin Administration*
Rapaport & Miller (19)	—	2-20	—
	—	3-20	—
	—	2.5-30	—
Bakhsh et al. (20)	—	1-25(8.2±4.2)	—
	—	3-24(9.1±3.6)	—
Banerjee & Guha (21)	1-2.52(2)	1.5	700 mgm. P.O. daily for 4 days
Glatt (22)	—	9-46	—
	?	13-40	½ Cebion tablet or 10-20 Gm. Zitronsaft t.i.d.
Rossini & Ferrari (23)	9-14	6-9	300-500 mgm. IV load dose
Uzan (24)	?	?	—
Uzan & Bronstein (25, 26)	?	?	—
Reddy & Sastry (27)	—	3.7-11.5(6.3)	—
	—	5.1-14.3(8.1)	—
	—	5.5-14.0(8.6)	—
Reddy & Sastry (28)	—	3-19(8.1)	—
	—	3.3-16.2(8.5)	—
	—	3-23.3(12.5)	—
Dollé (29)	?	?	
	?	?	
	15-45	4-10	Cebion 3 tablets b.i.d.
	35	15	Canton Forte (?)
	2-25	4-20	Cebion 3tablets b.i.d.
	17-46	8-28	Canton Forte (?)
	6-45	4-47	Ce Ferro (IV)
Slobody (30)	—	3.5-25.6	—
	?	over 14	9 week scorbutic diet
Banerjee (32)	5-12.5	2.7-5.5	200 mgm. daily per os
Gounelle & Vallette (33)	—	2-12	—
	—	2.5-13	—
	—	3-6	—
Moena & Nolff (34)	—	57% below 14 min. 77% below 17 min. 21% between 17-20 min. 2% between 21-25 min.	—
	—	39.8% below 14 min.	—
	over 14 min.	below 14 min.	oral or IV ascorbic acid. Dosage?
	19.7	16.4	
	11.2	9	
	16	16	500 mgm. P.O. per day
Zanotti (36)	—	20% between 10-15 50% between 15-25 30% over 25	—
Viñas-Espin (37)	—	?	—

.8 mgm. percent. Moena and Nolff then determined 24 hour urinary concentrations of ascorbic acid. They found a great periodic variation in the amount excreted during the day. They cautioned that single determinations are not sufficient to fix a diagnosis of vitamin C deficiency. The researchers concluded that treatment with vitamin C should be evaluated on a three-fold basis: (1) enhanced urinary ascorbic acid excretion, (2) 3 day normal fasting plasma level, and (3) normal skin time. Of these, the authors seemed to prefer the skin test.

BICKNELL AND PRESCOTT

Bicknell and Prescott,[35] having reviewed the literature, considered the intradermal test of no clinical value. They believed that too many variables exist to allow any specific knowledge of vitamin C status from the skin test time (other reducing agents, changes in temperature, circulation and oxygen supply).

ZANOTTI

Zanotti[36] employed Rotter's method to determine intradermal times in 50 frostbitten soldiers. All decolorization times were in excess of 10 minutes: 10 subjects ranged between 10 and 15 minutes; 25 yielded times between 15 and 25 minutes; 15 revealed skin times in excess of 25 minutes. The author felt that subnutritional states and frostbite are somehow related. For this reason Zanotti suggested that the test might measure only skin saturation which was not a true barometer of total body status. He gave large doses to the soldiers and found that the intradermal times diminished. He was then inclined to postulate that, perhaps, the Rotter skin time could be utilized in ascertaining complete saturation after prolonged ascorbic acid therapy.

VINAS-ESPIN

Viñas-Espin,[37] using a concentration of 40 mgm./100 cc. of DCPIP (approximately N/300) and an intradermal injection of .025 cc., found a clear decrease of decolorization time following the administration of 300 mgm. of ascorbic acid. Precisely how the ascorbic acid was given and the number and health of the patients was not mentioned. In addition, he determined the concentration and the absolute amount of ascorbic acid in the urine of patients who were given ascorbic acid and thiamin for 4 to 13 days. According to his observations, the first effect of vitamin C administration is a lowering of urinary ascorbic acid and phosphate. On the basis of this observation, Viñas-Espin postulated that ascorbic acid plays a role in phosphorylation. He found that thiamin hydrochloride, given after ascorbic acid had been taken regularly, further improved the cellular reductive power. Expressed more simply, the irreducible intradermal time following ascorbic acid intake can be further reduced with thiamin hydrochloride. Additionally, when thiamin was administered together with ascorbic acid, there was an increase in the urinary excretion of vitamin C. From these observations the author then theorized a relationship between the oxidative metabolism of tissue ascorbic acid and the phosphorylation process.

TABLE 4

Author(s)	*Purpose*
Rotter (2, 3)	To note whether changing body ascorbic acid levels affect the disappearance time.
Portnoy & Wilkinson (4, 5)	To relate skin test time to 5 other laboratory tests of vitamin C status.
Poncher & Stubenrauch (6)	To correlate skin test times with plasma ascorbic acid levels.
Jetter (7)	To correlate fasting whole blood values with skin times.

TABLE 4 (Continued)

Author(s)	*Purpose*
Codvelle et al. (8)	To establish optimal daily requirement of ascorbic acid as gauge of body status.
Gambigliani-Zoccoli & Lombardo (9)	To evaluate skin test.
Oliveira Lima (10)	To relate intradermal time to body saturation using urinary excretion as index.
Poulsen & Lieck (11)	To evaluate skin test.
Wright & MacLenathen (12)	1) To relate diet and blood ascorbic acid values to skin test time. 2) To relate site of injection to intradermal score.
Banerjee & Guha (13)	To relate decolorization time to urine ascorbic acid excretion after load dose.
Suzuki (14)	1) To relate the skin time to plasma ascorbic acid level. 2) To determine effect of diseased skin on DCPIP disappearance time.
Goldsmith et al. (15)	To correlate fasting plasma ascorbic acid values with skin scores.
Slavich & Torrini (16)	To evaluate skin test.
Baraldi (17)	To evaluate Rotter's test.
Beck & Krieger (18)	To evaluate skin test.
Rapaport & Miller (19)	To relate decolorization time to plasma ascorbic acid values.
Bakhsh et al. (20)	To correlate plasma levels and intradermal time.
Banerjee & Guha (21)	To determine a minimal intradermal time.
Glatt (22)	To correlate skin time with plasma level.
Rossini & Ferrari (23)	To evaluate skin time.
Uzan (24)	To evaluate intradermal test.
Uzan & Bronstein (25, 26)	To evaluate and improve Rotter test.
Reddy & Sastry (27)	To correlate intradermal time to amount of citrus intake.
Reddy & Sastry (28)	To compare skin test results of patients with various ophthalmic disorders.
Dollé (29)	1) To relate skin times to diet and medical history. 2) To relate skin times to Rumpel-Leede test.
Slobody (30)	1) To uncover a wheal size and dye concentration which will yield constant times. 2) To correlate plasma level with skin time.
Banerjee (32)	1) To study variations of skin time at different and same sites on consecutive days. 2) To determine the amount of ascorbic acid required for saturation. 3) To describe seasonal variation.
Gounelle & Vallette (33)	To correlate intradermal time with whole blood ascorbic acid.
Moena & Nolff (34)	To compare skin times in healthy and sick persons in a study of epidemiologic proportion.
Zanotti (36)	To determine ascorbic acid status of patients with frostbite by the intradermal test.
Viñas-Espin (37)	To observe whether the skin time is related to oxidation-reduction capacity of tissue.

TABLE 5

Author(s)	*Conclusions*	*Basis*
Rotter (2, 3)	Decolorization time depends on amount of ascorbic acid in or administered to the organism.	Skin times increased in persons on a 10 day scorbutic diet; they diminished after IV or oral vitamin administration.
Portnoy & Wilkinson (4, 5)	Times less than 5 minutes indicate saturation; over 10 minutes points to deficiency.	There was a close relationship of mean group skin time to ascorbic acid in diet and to the 5 other test scores indicative of body status.
Poncher & Stubenrauch (6)	Skin test is clinically unsatisfactory.	The plasma level and intradermal time were unrelated.
Jetter (7)	Skin test is non-specific.	There was no relationship between blood ascorbic acid and decolorization time.
Codvelle et al. (8)	100 mgm. per day is an optimal intake of ascorbic acid.	Persons having no supplementary vitamin C usually yielded skin times above 10 minutes. Those receiving a 100 mgm. addition showed times less than 10 minutes.
Gambigliani-Zoccoli & Lombardo (9)	Skin test is not useful when viewed in isolation.	As reported in reference,[1] they observed that guinea pigs on a deficient diet showed no increase in test time.
Oliveira Lima (10)	Skin test offers no utility.	Groups, demarcated by a urinary saturation test, displayed widely divergent skin times.
Poulsen & Lieck (11)	Skin test is not an estimate of tissue ascorbic acid concentration.	Skin time was not related to plasma level; it showed extensive variation with location, environmental temperature and blood flow.
Wright & MacLenathen (12)	Skin decolorization time is non-specific.	1) There was no correlation of DCPIP scores with diet and blood ascorbic acid concentration. 2) Times varied widely on same and distant sites.
Banerjee & Guha (13)	Skin time is qualitatively related to urinary vitamin C excretion.	The test time decreased as urinary ascorbic acid increased.
Suzuki (14)	1) Intradermal test is useful. 2) Intradermal times are shorter in borderline and diseased skin than in normal skin of the same subject.	1) Skin test times correlated well with plasma levels. 2) Author explained this phenomenon as a local increase in ascorbic acid due to inflammation.
Goldsmith et al. (15)	Intradermal test is worthless.	No correlation existed between plasma level and skin time.
Slavich & Torrini (16)	Skin test is a good, simple procedure.	Ill persons showed increased skin times.
Baraldi (17)	Clinically, the skin test is unsound.	The test did not distinguish between poorly nourished persons and those on a normal diet.
Beck & Krieger (18)	Skin test is a useful screening tool.	Test time was related to amount of ascorbic acid needed to effect urinary saturation. Persons having diseases associated with vitamin C deficiency showed longer times than other ill ones.
Rapaport & Miller (19)	Skin test is not satisfactory.	No one intradermal time or range was consistent enough to allow determination of individual status.
Bakhsh et al. (20)	Skin test is of no clinical value.	Although the two scores were not independent of each other, the wide individual variation allowed no critical analysis.

TABLE 5 (Continued)

Author(s)	*Conclusions*	*Basis*
Banerjee & Guha (21)	The minimal intradermal time is 90 seconds.	Human beings yielded decolorization times of 90 seconds after 4 days of 700 mgm. ascorbic acid daily.
Glatt (22)	Skin time has no clinical value.	No significant difference was observed between supplemented and non-supplemented groups.
Rossini & Ferrari (23)	Intradermal test is not practical.	Tubercular patients, shown to be deficient by capillary fragility and urinary saturation, did not exhibit prolonged times.
Uzan (24)	The Rotter test is a valuable clinical adjunct.	A parallel was evident between skin time and urine saturation test.
Uzan & Bronstein (25, 26)	1) Times of 2-3 minutes indicate saturation; over 10 minutes deficiency. 2) Substitute methylene blue for DCPIP.	1) No results were given in available publications. 2) Methylene blue was more specific for vitamin C than DCPIP at the normal dermal pH.
Reddy & Sastry (27)	Skin time is reliable index of tissue ascorbic acid status.	Children and young adults on good diets exhibited times below 10 minutes.
Reddy & Sastry (28)	Ascorbic acid deficiency accompanies inflammatory eye conditions.	Patients having ophthalmic inflammation yielded prolonged intradermal times.
Dollé (29)	1) Intradermal test is good rough index of individual status. 2) Rumpel-Leede test may be poor clinical method.	1) Patients with heaviest blood loss showed longest times. Skin times tended to parallel diet. 2) There was no correlation between the capillary fragility and intradermal scores.
Slobody (30)	1) N/300 dye solution and a 4 mm. wheal are best. 2) Skin test time and plasma levels are not related. 3) Intradermal test measures tissue ascorbic acid level.	1) This combination yielded most constant times. 2) Author felt that plasma levels reflected only recent intake. 3) Skin test times seemed to follow biomicroscopic findings of a scorbutic character in the gingivae.
Banerjee (32)	1) Skin test serves only in group evaluation. 2) 200 mgm. ascorbic acid daily cannot saturate body. 3) Skin test times are lower in February than September.	1) Individual differences were extensive. There were significant differences within same individual on consecutive days. 2) A previously established minimal time of 90 seconds was not attained after 3 weeks of a 200 mgm. daily dose. 3) Fresh fruits and vegetables were not available in September.
Gounelle & Vallette (33)	Intradermal time cannot replace blood determination.	There was no correlation between skin test time and whole blood ascorbic acid.
Moena & Nolff (34)	1) Ill persons have lowered ascorbic acid reserves. 2) Skin test time is better than plasma level as clinical procedure.	1) Increased times were more frequent in diseased persons. 2) Plasma levels showed wide variations in short time. Skin times changed only slowly.
Zanotti (36)	Skin test is not satisfactory.	A wide range of skin times was found in soldiers who had been on the same diet for months. The diet was deficient in ascorbic acid; yet the intradermal times, in general, were not greatly prolonged.
Viñas-Espin (37)	The Rotter time reflects the oxidation-reduction capacity of the skin.	The enhancement of the phosphorylation process by administration of thiamin paralleled a decrease in the skin time.

SUMMARY

The following tables are submitted as a historical outline of the intradermal ascorbic acid test in human subjects. All reports available to the compilators were based on the use of 2,6 dichlorophenolindophenol. From Table 1 it is evident that: (1) of those who report the quantity of dye injected, all investigators except four utilized a .01 cc. injection as proposed by Rotter, (2) a DCPIP concentration of N/400 was employed in all studies except six, (3) when wheal size was specified, it was, with three exceptions, 2 mm., and (4) the forearm was the most popular injection site.

Table 2 summarizes the number and health of the subjects and the length of the investigation.

Table 3 outlines the essential experimental data relative to the intradermal test. Examination of the tabulation reveals: (1) in only 11 out of 31 investigations are there scores reported both at the start and finish of the investigation, (2) groups of subjects deficient in ascorbic acid generally require a longer therapeutic interval to reduce the DCPIP disappearance time, (3) group administration of ascorbic acid is followed by a diminution in decolorization time—and the decline is related to the amount of vitamin given, the route of administration, and the time interval between dosage and final intradermal testing.

The essential aim of each report is briefly stated in Table 4. The purpose may be summed up in a few statements: (1) to ascertain whether the decolorization time varies with decrease and increase of body ascorbic acid, (2) to establish whether a significant relationship exists between the intradermal score and other measures of vitamin C status—particularly the fasting plasma ascorbic acid concentration, (3) to evolve an irreducible skin test value indicative of absolute saturation, (4) to determine local variables affecting the final decolorization score, (5) to establish empirically a dye concentration and wheal size which provide repetitive decolorization times, (6) to relate cutaneous decolorization scores to age, (7) to contrast the skin test values of healthy and ill subjects, and (8) to disclose whether the Rotter test measures oxidation-reduction capacity of the tissues.

The evidence from Table 5 is that: (1) the decolorization time does vary inversely with changes in total body ascorbic acid, (2) there is no experimental unanimity concerning the presence or absence of a relationship between Rotter's time and plasma ascorbic acid content, (3) ninety seconds is an irreducible DCPIP disappearance time, (4) changes in age and concentration of solution, vascular flow, site of injection, wheal size, light intensity, and depth of puncture do significantly alter the decolorization time, (5) a dye concentration of N/300 and a wheal size of 4 mm. yields the most reproducible results, (6) skin test time appears to increase with age and/or disease, (7) the cutaneous vanishing time varies with changes in oxidation potential of the tissues.

A subsequent publication[88] will concern itself with an analysis of a group of our own patients.

University of Alabama School of Dentistry,
Birmingham, Alabama

BIBLIOGRAPHY

1. Cheraskin, E., Dunbar, J. B. and Flynn, F. H. The intradermal ascorbic acid test. Part I. A review of animal studies. Jour. Dent. Med. 12:174-184, Oct. 1957.
2. Rotter, H. Determination of vitamin C in the living organism. Nature 139: #3521, 717, April 24, 1937.
3. Rotter, H. Bestimmung des vitamin C in lebenden organismus. Wiener Klin. Wchnschr. 51: #7, 205-206, February 18, 1938.
4. Portnoy, B. and Wilkinson, J. F. Intradermal test for vitamin C deficiency. Brit. Med. Jour. 1: #4023, 328-329, February 12, 1938.
5. Portnoy, B. and Wilkinson, J. F. Vitamin C deficiency in peptic ulcer and haematemesis. Brit. Med. Jour. 1: #4027, 554-560, March 12, 1938.
6. Poncher, H. G. and Stubenrauch, C. H. Intradermal dye test for vitamin C deficiency. J. A. M. A. 3: #4, 302-304, July 23, 1938.
7. Jetter, W. W. Correlation between blood ascorbic acid and the dichlorophenolindophenol intradermal test. Proc. Soc. Exper. Biol. and Med. 39: #1, 168-171, October 1938.
8. Codvelle, E., Simonnet, H. and Mornard, J. Recherches sur la carence en acide ascorbique. Presse Med. 46: #95, 1745-1748, November 26, 1938.
9. Gambigliani-Zoccoli, A. and Lombardo, V. L'intradermoreazione di Rotter quale indice del grado di saturazione dell'organismo in vitamina C. Riforma Medica 54: #39, 1489-1490, October 1938; Accademia di Med. di Torino Gior. 101: #3, 428-433, July-September 1938.
10. Oliveira Lima, A. Sobre o valor do "test" intradermico com o dichlorophenol para o reconhecimento de estados sub-escorbuticos. Brasil-Medico 53: #5, 178-180, January 1939.
11. Poulsen, E. and Lieck, H. Undersolgelser over H. Rotter's intracutanprove paa mennesker til paavis-

ning of vitamin C. Ungeskrift for Laeger 101: #7, 206-208, February 16, 1939.
12. Wright, I. S. and MacLenathen, E. The intradermal test for vitamin C determination. Jour. Lab. and Clin. Med. 24: #8, 806-807, May 1939.
13. Banerjee, S. and Guha, B. C. The intradermal test as an index of vitamin C nutrition. Part II. Indian Med. Gaz. 75: #8, 468-471, August 1940.
14. Suzuki, S. Vitamin C and skin diseases. Report II. Intradermal test with 2,6 dichlorophenolindophenol. The Jour. of Oriental Med. 30: #6, 267-269, June 1939.
15. Goldsmith, G. A., Gowe, D. F. and Ogaard, A. T. Determination of vitamin C nutrition by means of a skin test. A critical evaluation. Proc. Soc. Exper. Biol. and Med. 41: #2, 370-374, June 1939.
16. Slavich, E. and Torrini, A. Prova di Rotter e suoi rapporti con il contenuto cutaneo di vitamina C. Gior. di Clin. Med. 20: #9, 831-841, June 30, 1939.
17. Baraldi, M. La vitamina C determinata col metodo di Rotter nell'organismo dei malati di mente. Revista Sperimentali di Freniatria 63:368-373, June 30, 1939.
18. Beck, H. and Krieger, F. H. Was leistet Eine Intrakutanprobe mit Indophenol zur Prufung des Vitamin C- Haushaltes? Deut. Med. Wchnschr. 65: #34, 1336-1340, August 1939.
19. Rapaport, H. G. and Miller, S. H. The determination of vitamin C in children by intradermal injections. Jour. Pediat. 15: #4, 503-507, October 1939.
20. Bakhsh, M. I., Kochlar, B. D. and Malik, A. W. The evaluation of the intradermal dye test for vitamin C in health and disease. Indian Jour. Med. Res. 27: #3, 695-703, January 1940.
21. Banerjee, S. and Guha, B. C. The intradermal test as an index of vitamin C nutrition. Part IV. The relation between the intradermal test time and the ascorbic acid content of the liver of guinea pigs. Ann. Biochem. and Exper. Med. 2: #2, 121-124, March 1942.
22. Glatt, J. Die Beurteilung der Vitamin C. Sattigung der Gewebe durch die Intrakutanreaction von Rotter bei Kranken und rekonvaleszenten Sauglingen. Dent. Med. Wchnschr. 66: #47, 1304-1305, November 1940.
23. Rossini, G. and Ferrari, B. Sul valore della intradermoreazione di Rotter nella tuberculosi pulmonare. Boll. Soc. Med.-Chir. Pavia 54: #6, 1277-1285, November-December 1940.
24. Uzan, M. L'appreciation de l'hypovitaminose C, par le test de Rotter. Medecin chez Lui 15: #14, 13-14, July 1938.
25. Uzan, M. and Bronstein, I. Epreuve de Rotter et ascorbotherapie. Abstracted in: Zeitschrift f. Vitaminforschung 9: 379-380, 1939.
26. Uzan, M. and Bronstein, I. Contribution nouvelle a l'etude de l'epreuve de Rotter. Medicin chez Lui 16: #9, 16-21, May 1939.
27. Reddy, D. V. S. and Sastry, P. B. Intradermal test as an index of vitamin C nutrition of the body—observations at Vizagapatam. Indian Med. Gaz. 76: #8, 476-480, August 1941.
28. Reddy, D. V. S. and Sastry, P. B. Intradermal test for vitamin C nutrition of the body in ophthalmic patients. Jour. Indian Med. Assn. 10: #11, 440-443, August 1941.
29. Dollé, W. Uber Vitamin-C-Schnellbestimmung mit dem intradermalen Testverfahren nach Rotter. Zentralbl. f. Gynak. 65: #8, 1414-1418, August 2, 1941.
30. Slobody, L. B. An intradermal test for vitamin C subnutrition. Preliminary report. Jour. Lab. and Clin. Med. 29: #5, 464-472, May 1944.
31. Kruse, H. D. The gingival manifestations of avitaminosis C, with especial consideration of the detection of early changes by biomicroscopy. Milbank Mem. Fund Quarterly 20: #3, 290-323, July 1942.
32. Banerjee, S. A study of the intradermal test of Rotter for vitamin C status. Indian Med. Gaz. 79: #9, 420-423, September 1944.
33. Gounelle, H. and Vallette, A. Test de Rotter et teneur en vitamine C. Comp. Rend. d. Sean. de la Soc. de Biol. 138: #19-20, 740-742, October 1944.
34. Moena, A. and Nolff, E. Test intradermico para la determinacion de la saturacion tisular en vitamina C. Anales Medicos de Conception 2: #3 & 4, June-December 1945.
35. Bicknell, F. and Prescott, F. The vitamins in medicine. Ed. 2., 1946. New York, Grune and Stratton. pp. 542-543.
36. Zanotti, M. Risultati della intradermoreazione di Rotter nei militari colpiti da congelamento. Gior. Med. Mil. 94: #6, 442-453, November-December 1947.
37. Viñas-Espin, J. Le test de Rotter dans la determination de l'avitaminose C-B1 chez l'homme. Bull. Soc. Chim. Biol. 33: #9, 1168-1173, Summer 1951.
38. Cheraskin, E., Dunbar, J. B. and Flynn, F. H. The intradermal ascorbic acid test. Part III. A study of 100 patients. (To be published).

LE TEST INTRADERMAL A ACIDO ASCORBIC PARTE II. UN REVISTA DE STUDIOS IN HUMANOS

Dr. med. dent. J. B. Dunbar, Dr. med. e med. dent. E. Cheraskin, e F. H. Flynn (B.S., M.T., A.S.C.P.)

SUMMARIO IN INTERLINGUA

Le sequente tabulas es offerite como un delineation historic del test intradermal a acido ascorbic in subjectos human. Omne le reportos accessibile al compilatores esseva basate super le uso de 2,6-dichlorophenolindophenol. Tabula 1 rende evidente que (1) inter omne le investigatores reportante le quantitate del colorante injicite, solmente quatro non sequeva le recommendation de Rotter de utilisar injectiones de 0,01 cm^3, (2) un concentration de N/400 esseva usate in omne le studios con sex exceptiones, (3) quando le dimensiones del urticas esseva indicate, illos esseva 2 mm, con tres exceptiones, e (4) le antebracio esseva le plus frequente sito del injection.

Tabula 2 summarisa le numero e le stato de sanitate del subjectos e le duration del investigationes.

Tabula 3 delinea le importante datos experimental relative al test intradermal. Le examine del tabula monstra que (1) il es in solmente 11 inter 31 investigationes que valores es notate tanto al comenciamento como etiam al fin del investigation, (2) subjectos a carentia de acido ascorbic require generalmente un plus longe intervallo therapeutic pro effectuar un reduction del tempore de disparition de dichlorophenolindophenol, e (3) in gruppos recipiente administrationes de acido ascorbic il seque un reduction del tempore de discoloration, e iste reduction es relationate al quantitate de vitamina administrate, al via de administration, e al intervallo de tempore inter le dosage e le ultime test intradermal.

Le objectivos essential del varie reportos es summarisate brevemente in Tabula 4. Iste objectivos pote esser concentrate in le sequente formulationes. Il se tractava (1) de determinar si le tempore de discoloration varia con le reduction e le augmento del acido ascorbic in le corpore, (2) de establir si il existe un relation significative inter le valor intradermal e altere mesuras del stato de vitamina C, specialmente le valor de acido ascorbic del plasma in stato jejun, (3) de evolver un irreducibile valor de test cutanee que indicarea un saturation absolute, (4) de determinar variabiles local que affice le ultime valor pro le discoloration, (5) de establir empiricamente un concentration del colorante e un dimension del urtica que provide repetitive tempores de discoloration, (6) de relationar valores de discoloration cutanee al etate del subjectos, (7) de contrastar le valores del test cutanee in subjectos pathologic e normal, e (8) de constatar si o non le test de Rotter mesura capacitates de oxydation e reduction del histos.

Le factos que pote esser legite in Tabula 5 es que (1) le tempore de discoloration varia inversemente con alterationes del total acido ascorbic del corpore, (2) il non existe un accordo experimental relative al presentia o absentia de un relation inter le tempore de Rotter e le contento de acido ascorbic in le plasma, (3) novanta secundas es un irreducibile tempore pro le disparition de dichlorophenolindophenol, (4) alterationes de etate e del concentration del solution, le fluxo vascular, le sito del injection, le dimensiones del urticas, le intensitate de lumine, e le profundor del punctura altera significativemente le tempore del discoloration, (5) un concentration del colorante de N/300 e un dimension del urtica de 4 mm es associate con le plus alte grado de reproducibilitate del resultatos, (6) il pare que le tempores del test cutanee cresce con le etate e/o le presentia de morbo, e (7) le tempore de disparition cutanee varia con alterationes del potential de oxydation in le histos.

Case Report

ENDODONTIA FOR CUTANEOUS LESIONS OF DENTAL ORIGIN

MORRIS M. GLASSER, D.D.S., F.A.C.D.

Mead[1] states "that a fistula differs from a sinus in being open at both ends. It is an abnormal communication, therefore, between two surfaces lined with epithelium. Occasionally a fistula results from ulceration of a malignant growth."

Montgomery[2] describes several cases of dental fistulas that simulated dermal epitheliomas. Wende and Solomon[3] in 1942 reported a case that proved to be a squamous cell carcinoma superimposed upon a chronic dental sinus. Gurdin and Pangman[4] in 1953 reporting on Dermal Sinuses of Dental Origin, describe the possible mistaken diagnosis of these cutaneous lesions for epidermoid cysts.

During the course of pulpal disease, the condition can erupt as an acute alveolar abscess or a chronic alveolar abscess. In the acute stage, seepage of toxic protein or infective bacteria from the necrotic pulp through the apical foramen will cause a severe reaction. This inflammatory response by the tissue may produce pus within 24-48 hours which may escape through the tissues or avenues of least resistance. This escape area when above the offending tooth is known as a parulus or gum boil. Unless this parulus develops spontaneously, incision for drainage is paramount so as to avoid concomitant systemic reactions of a very serious nature. X-rays may reveal no apparent osseous pathology in the acute phase.

Most abscesses become circumscribed and contained within the alveolar framework of the root. When the irritating factors produce pus, this dissects the subperiosteum and attempts to evacuate itself into the soft tissues. Macomber, Wang and Gottlieb[5] tabulate at length the possible places these fistulous tracts may terminate. Stones[6] even describes the closed spaces of the neck that may become involved producing serious infections. With the advent of chemotherapeutic agents and antibiotics, such serious involvements are seen less and less.

A. Intra-Orally:
   1. The fistula may be on the buccal or labial side directly over the apex or nearby the root-end
   2. Back through the root canal into the pulp chamber; if the cavity is open to allow such a back flow
   3. Laterally into the gingival sulcus
   4. Palatally
   5. Antrally—when the roots adjoin the floor of the antrum
   6. On occasion into the floor of the nose

B. Cutaneously:

   A cutaneous lesion is influenced by the location of the abscess. The lower anterior teeth will tend to drain to the chin, neck and submaxillary region. The maxillary teeth will cause external fistulas beneath the zygoma and even the inner canthus of the eye.

The presence of an ulcerating cutaneous lesion on the face discharging purulent material could quite naturally cause the patient to seek medical help. Unless the physician is alert to the dental problems involved, a wasteful and prolonged treatment of this lesion will not only be without result, but also could produce deforming scar tissue with invagination of the skin. The usual efforts to incise and enucleate this apparent nodule of infection by the medical surgeon can only lead to frustration with recurrence of the ulcer.

Sommer[7] feels that negative bacteriological cultures will be possible when the fistulous tract is eliminated first. He recommends that a "fine, blunt explorer dipped into iodized phenol" be

* Instructor in Endodontics, Tufts University School of Dental Medicine.

passed into the fistulous tract to cauterize the epithelial lining of the tract. This should close the fistula long enough for the microorganisms to be destroyed by the tissue cells, then a negative culture can be obtained from the root canal and the canal obturated permanently. Sommer continues to reaffirm the newer concept of periapcal lesions, namely, that surgery is not always necessary. Bone repair of these radiolucent lesions about the root apex will occur by the natural process of tissue repair once the irritating factors have been removed or reduced and brought within the physiological limits of tissue tolerance.

The external ulcer will heal rapidly with no further medical treatment needed. Should the scar tissue still be prominent, a plastic correction may be required. It must be stressed, however, that plastic surgery be delayed until the underlying connective tissue beneath the derma has fully matured.

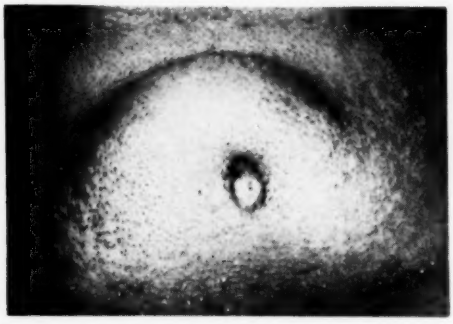

Fig. 1. Cutaneous Lesion of chin: Showing hypertrophied mass with central lumen of fistula through which exudate is continuously discharging. Hair follicles are secondarily involved by constant irritation and crustration of discharge.

CASE REPORT

J.Z., a 35 year old male, presented an ulcerative discharging nodule on the chin. (Fig. 1.) No dental pain or discomfort prompted his seeking further treatment except that the invagination on the chin prevented him from shaving. The continuous drainage also began to crustate around the hair follicles presenting an unsightly appearance. (Fig. 1.)

CASE HISTORY

His dental history revealed that 7 years ago he had a swelling of his lower lip in the region of the lower central incisors with accompanying pain. Dental roentgenograms revealed no bone pathology nor was caries of the teeth present. However, tenderness on percussion of the incisors was affirmed. Subjective symptoms of a sub-acute abscess seem to have been present. However, no specific dental treatment was instituted other than placing a poultice on the swelling in the mouth. The swelling regressed and the patient became comfortable. Thereafter, a small area appeared on the chin which began as a small boil. This ruptured and an exudate discharged.

Fig. 2. Cutaneous Lesion on Chin: Showing regression of lesion. The root canal has been biomechanically prepared, sterilized and obturated so that periapical repair will occur by the physiological processes of the body. This is a normal response when irritating factors have been brought within the physiologic limits of tissue tolerance.

MEDICAL THERAPY

Medical treatment was started by his physician in the following order:

1. Salves to be applied to the face
2. Irrigations by the physician
3. Heat in the form of hot packs and poultices
4. Antibiotics
5. Surgery-excision and drainage. No particular relief was obtained except that after the surgery the skin invaginated.

Fig. 3

Fig. 4

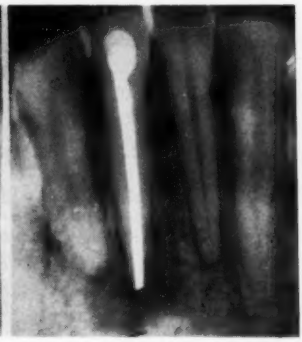

Fig. 5

Fig. 3. Pre-operative X-rays: The periapical pathology appears to involve the left central and lateral incisor. Actually it was the lateral incisor. Pulp test of the central was normal.

Fig. 4. Hypaque, a radio-opaque fluid has been forced through the root canal, into the bone lesion and through the fistulous tract coming out on the external opening of the cutaneous lesion.

Fig. 5. Root Canal Completed: The canal has been obturated with a silver point and sealer. The periapical lucency will heal in due time with new bone deposit. The cutaneous lesion has healed completely.

The patient then consulted a dentist. In the course of his regular dental examination, a radiograph revealed periapical pathosis about the apices of the lower central and lateral incisors. (Fig. 3.) A consultation was arranged with the dental oral surgeon who advised against extraction of these teeth, but advised root canal therapy and further correlation of these diseased teeth with the external fistula.

CLINICAL EXAMINATION

Various endodontic tests were utilized which pointed to the central incisor as the offending tooth. Whether the lateral incisor was also a contributing cause to this bone lesion was kept in abeyance until further radiographic examination could limit the lesion.

ROENTGENOGRAPHIC EXAMINATION AND INSTILLATION OF RADIO-OPAQUE SOLUTION

Regular dental examination revealed a bone lucency about the apices of the lower left central and lateral incisors. (Fig. 3.) Once the root canal was made patent, an antiseptic solution was forced through the root canal into the osseous bone through the labial cortical plate of bone, thence through the fistulous tract to the external parulus of the chin. The radio-opaque fluid was then instilled in a similar manner and radiographs taken. (Fig. 4.) The extent of the bone lesion is thus outlined about the apex of the central incisor. (Fig. 4.)

ENDODONTIC OPERATION

Regular root canal therapy was instituted in the following order:

1. Pulp chamber opened on the inner surface of the tooth
2. Canal reamed and filed to the apex and lavaged
3. Canal filling prepared
4. Medication of the canal with bacteriacide
5. Bacteriological cultures taken
6. Canal obturated. (Fig. 5.)

POST OPERATIVE SURVEY

The external fistula responded by healing rapidly with diminishing exudate until no further discharge appeared. (Fig. 2.) The connective tissue beneath the skin began to repair the deformity and the derma took a more nat-

ural contour. A very slight indent remained but in no way interfered with shaving. No other tooth was involved in this pathology. Healing should take place in due time by a redeposit of calcium salts within the new bone lamela being laid down by osteogenic repair.

CONCLUSIONS

Diseased pulps can be the cause of many ills other than contained periapical lesions. When the diseased nerve and its adnexa revert to a chronic state of inflammation, a parulus may develop which can simulate an independent lesion or neoplasm. With the advent of newer methods in root canal therapy, a more specific diagnosis of root end pathosis can now be made and possible surgery and extractions may be avoided.

311 Commonwealth Avenue
Boston 15, Mass.

BIBLIOGRAPHY

1. Mead, Sterling V.; Oral Surgery, C. V. Mosby Co., St. Louis, 1940.
2. Montgomery, D. W.: Fistulas from Dead Teeth Simulating Dermal Epitheliomas, Arch. of Derm. and Syph., 41; 378-379, Feb. 1940.
3. Wende, R. C. and Solomon, H. A.: Cutaneous Fistulas of Dental Origin, Arch. of Derm. and Syph. 46; 665-672, Nov. 1942.
4. Gurdin, M. and Pangman, W. J.: Dermal Sinuses of Dental Origin. Plastic and Recon. Surg., 2: 444-452, June, 1953.
5. Macomber, W. B., Wang, M. K. H. and Gottlieb, E.; G. P. 14:81 Nov. 1956.
6. Stones, Hurbert H.: Oral Dental Diseases, E. & S. Livingstone, Ltd., Edinburgh and London, 3rd Edition, 1954.
7. Sommer, Ostrander, Crowley: Clinical Endodontics, W. B. Saunders Co., Philadelpria, 1956.

THE AMERICAN INSTITUTE OF DENTAL MEDICINE CASE HISTORY SERVICE

The Institute continues to offer Case Histories from the broad field of Dental Medicine for the year 1957-58. These will include original 35 mm. Kodachrome slides of the oral condition, pertinent laboratory findings, medical background, roentgenograms, photomicrographs, diagnostic criteria and whatever else may be indicated in each individual case, with a "general discussion" of the disease. A loose leaf binder and matching Kodachrome slide box will be furnished to each participant. The fiscal year of the Institute runs from October 1 to September 30.

Anyone subscribing to this Case History Service is entitled to full membership in The American Institute of Dental Medicine. Members of the A.D.A. or A.M.A., or their foreign equivalent, are eligible to apply. Due to the great demand for this service, it is recommended to subscribe at an early date for immediate delivery.

For further information, address all communications to the Secretary, Mrs. C. Novembri, 2240 Channing Way, Berkeley 4, California.

Medical Views on Oral Problems

DRYNESS OF MOUTH AND TONGUE*

TO THE EDITOR:—*A patient, aged 70 years, has had an absolute dryness of the mouth and tongue for the last two years. Examination is negative except for moderate obesity and associated hypertension, which has been corrected, and mild conjunctivitis. The salivary glands are not unusual. Complete blood cell count, urinalysis, nonprotein nitrogen level, blood cholesterol level, and blood electrolyte concentrations are all within normal limits. Basal metabolic rate and radioiodine (I^{131}) uptake are normal. The patient is not a mouth breather. He states, however, that there are occasional periods in which the mucous membrane has a little moisture. What can be done to help this patient?*

J. Will Fleming Jr., M.D., Moberly, Mo.

This inquiry has been referred to two consultants, whose respective replies follow.—ED.

ANSWER.—It is possible, and perhaps likely, that the medication used for control of hypertension is responsible for the dryness of the mouth and tongue. This is a common complaint of patients who are receiving Rauwolfia-like preparations. Dry mucous membrane in the mouth is sometimes seen in association with the pharyngitis sicca which usually accompanies both true atrophic rhinitis and the atrophic type of chronic rhinitis often present in elderly people. The latter may evidence endocrine changes after the male or female climacteric. Checking hypertensive drugs used for this patient and the administration of potassium iodide, 0.3 Gm. three or four times daily, together with an adequate fluid intake, might help this patient.

ANSWER.—The condition described would seem to meet the requirements for classification as Sjögren's syndrome. According to Dorland, this syndrome is characterized by "dryness of all mucous membranes, resulting from deficient secretion of the glands, particularly the lacrimal and salvary glands, those of the upper respiratory tract, the sweat glands, and the glands of the stomach." The syndrome usually appears in elderly people. There is no known treatment which has a general effect. Locally, artifical tears can be used in the eye whenever the symptoms require. The most satisfactory of the artificial tears is Isopto-Alkaline Ophthalmic Solution. Cortisone applied locally to the eye has an ameliorating effect for some patients who have this syndrome, but when it is taken by mouth it is of no value.

* Queries and Minor Notes, J.A.M.A., Sept. 7, 1957, p. 115.

PREMATURE ERUPTION OF TEETH*

TO THE EDITOR:—*A dentist states that a 6½-year-old boy has "teeth that are coming in five years ahead of time" and that these teeth "have so many cavities that he will have no teeth left by the time he reaches adulthood." His deciduous teeth also showed many cavities, and many of them had to be extracted. The dentist feels that this indicates some systemic disease. Complete physical examination reveals a boy who is tall for his age and in excellent physical and mental health. There are no congenital abnormalities apparent nor any evidence of disease. Serum calcium, phosphorus, and alkaline phosphatase levels are within normal limits for his age. A careful nutritional and hygienic history indicates that the child eats well of all foods, including those containing calcium and proteins, meat as well as milk, and brushes his teeth frequently. He does not overindulge in bottled sodas, orange juice, or milk to the extent of spoiling his appetite for meat. His ancestry is mixed Irish, German, and English, so that, for the present, his Irish ancestors are being blamed for his dental difficulties. Please suggest further investigation, possible diagnosis, therapy, and prevention. If each decalcified area is filled as early as possible, is there any reason why the child should lose his permanent teeth any earlier than anyone else?*

Shepard G. Aronson, M.D., New York

This inquiry has been referred to three consultants, whose respective replies follow.—ED.

ANSWER.—Premature eruption of teeth may occur in hypergonadism, which may be secondary to hyperpituitary function or, more rarely, hyperthyroidism. The degree of premature tooth eruption cannot be evaluated, since this information is not available. Blood chemistry findings would not be altered in rampant caries. The diet should be well balanced and low in fermentable carbohydrates.

The patient's dentist should seek the services of a caries control laboratory. The relative susceptibility of this patient to caries and the effectiveness of the caries control procedures can be evaluated by means of counts of lactobacilli. With frequent expert dental care and complete patient cooperation, there is no reason why the permanent teeth should be lost prematurely. Dental caries are related primarily to local environment factors that can be controlled.

ANSWER.—To begin with, it must be borne in mind that there is extensive variation in the eruption pat-

tern of both deciduous and permanent teeth and that the statement that the teeth "are coming in five years ahead of time" in itself is almost impossible to interpret. The lack of any evidence of a systemic disease would strongly suggest that this child's problem is entirely local. There is every reason to believe that if the extensive caries are managed on the basis of individual cavities the child should be able to maintain many of his teeth well into adult life. The question as to whether or not each decalcified area should be filled as early as possible to avoid the loss of more permanent teeth is the essence of the problem. Certainly this procedure should be followed, and the results should be acceptable. It is recommended that good local dental care be given the child in order to prevent the spread of the carious lesions and to maintain the teeth which are now forming the dentition.

ANSWER.—Tooth eruption normally occurs over a wide age range around the accepted average. Hyperthyroidism and hyperpituitary disease are associated with accelerated tooth eruption. Caries resulting from systemic disease most often result from defective formation of enamel predisposing to bacterial erosion of the teeth. From the description this child has no significant systemic disease. The early loss of teeth will often result in minor degrees of malocclusion, which predisposes to food being trapped and permitting overgrowth of oral bacteria with enamel erosion and caries formation. The age-periods 5 to 8 years and 12 to 18 years are periods of an unusually high incidence of caries. The best management in such a case is good oral hygiene and early filling of cavities with close supervision by the dentist. If teeth become so badly diseased that they must be extracted, space retainers should be used to maintain proper occlusion. No accurate prognosis can be given, but with proper care the child should have functioning teeth for quite a while.

* Queries and Minor Notes, J.A.M.A., Sept. 7, 1957, p. 112.

MILK CONSUMPTION BY ADULTS*

TO THE EDITOR:—*A young man, aged 21 years, in good health, has never abandoned drinking milk with his meals. He dislikes coffee or tea and consumes at least one quart of milk daily. Should interdiction be suggested to prevent possible metabolic disturbances or the deposition of renal calculi as he grows older?*

Joseph Backlar, M.D., Richmond Heights, Mo.

This inquiry has been referred to two consultants, whose respective replies follow.—ED.

ANSWER.—Milk is an important source of nutrients which are needed throughout life, and its continued consumption by adults is desirable accordingly. There is no evidence justifying the interdiction of the drinking of one quart a day by a healthy adult on the basis that this use might cause metabolic disturbance or the deposition of renal calculi.

ANSWER.—The question pertaining to "potential metabolic disturbances" suggests cholesterol metabolism. This whole subject is still in the realm of active research. If fat intake seems most important in this connection, one should recognize that milk is by no means a high-fat food (when it contains 3.9% fat by weight, giving 51.3% of the total calories). On this basis milk would not seem to be the first food to be eliminated to reduce fat intake. If atherosclerosis were known to exist or if blood cholesterol levels were unusually high, the use of milk reduced in fat might be considered. This choice, however, may be contraindicated when one considers the question pertaining to kidney stones. Some evidence has been reported relating renal calculi to vitamin A deficiency in animals. If there were any question of diabetes or other metabolic disturbance interfering with the formation of vitamin A from carotene, the 1,600 I. U. or more of preformed vitamin A obtained from a quart of whole milk would be a safeguard. So far as the possibility that the one quart plus other intake of milk would make calcium intakes so high as to raise question of supercalcification is concerned, apparently even doubling the normal level of calcium intake has no such effect in the normal individual. Again, only if the patient already had evidence of renal calculi or belonged to a family where the incidence of stones is high would there seem to be any reason to anticipate problems related to the levels of calcium intake which are suggested by the ingestion of even more than one quart of milk daily. Until further evidence is available, it would seem reasonable to permit this normal young man to continue to enjoy his quart or more of milk per day.

* Queries and Minor Notes, J.A.M.A., Sept. 7, 1957, p. 113.

LOSS OF TOOTH ENAMEL*

TO THE EDITOR:—*A woman, 34 years old, is losing the enamel of her teeth, which are becoming discolored and painful. She is the mother of two children, aged 5 and 2 years, and has no significant history or symptoms. She wore an orthodontic brace on her teeth when she was a child. What can be done for her?*

Louis Gold, M.D., Lido Beach, L. I., N. Y.

ANSWER.—Discoloration and loss of tooth enamel in an adult is usually of local environmental origin, owing to the materials which contact the teeth. The main nocuous agents in this regard are usually (1) excessive intake of lemon or other foods containing citric or malic acid, (2) use of excessive amounts of refined starches, e. g., hard candies, cough drops, candy-coated chewing gum, sugar in coffee or tea, or sweetened, acidified carbonated beverages, or (3) acid gastric regurgitation. Many other factors, such as

change in pH or buffering ability of the saliva due to emotional tension, excessive drying from radiation involving the salivary glands or from mouth breathing, improper or inefficient tooth-brushing techniques, may be involved. In studying dietary intake, it is much better to have the patient keep a dietary diary for a full week rather than to rely on office questioning of the patient.

* Queries and Minor Notes, J.A.M.A., Sept. 14, 1957, p. 200.

BENIGN MASSETERIC HYPERTROPHY

The enlargement of the masseter muscle gives the appearance of a tumor above the angle of the mandible.

There are two types of this muscular hypertrophy. First, there is a congenital or familial type in which the muscle stands out because of a thin or bony face giving a "chipmunk" appearance.

Patients with the second type which is acquired, are typically tense and habitually clenchers or grinders of their teeth. The acquired condition is the type most often confronted by the dentist and physician.

On clinical examination, a malocclusion or wearing down of the teeth surfaces may be found. The syndrome may be unilateral or bilateral and may result in a lateral "flaring" of the mandible due to abnormal tension from the muscle. When the condition occurs unilaterally, the facial appearance and distortion of the mandible as shown by x-ray may be alarming. This symptom complex has been reported also in individuals who "cradle" their jaws with their hands while reading, displacing the mandible laterally.

A differential diagnosis must be made among patients suffering from this syndrome, and those with parotitis, parotid tumor, lipoma, rhabdomyoma, and even otitis externa (when associated with pain).

Proper therapy must include returning the occlusion to normal, eliminating any abnormal habits and to reassure the patient concerning a malignancy.

By Richard T. Barton, M.D.
Abstracted by Leonard M. Nevins, from J.A.M.A. Aug. 10, 1957, pgs. 1646-1647.

FLAVONOIDS IN HUMAN NUTRITION AND MEDICINE

The recent upsurge of interest in the flavonoids particularly in the use of these compounds in the treatment of numerous clinical disorders has prompted the compilation of this review.

The high hopes once held for the flavonoids as important metabolites have not materialized. Instead present knowledge indicates that while they possess mild pharmacological properties under certain conditions, the flavonoids have no known nutritional functions. They cannot be regarded as essential nutrients. Those workers who claim therapeutic value for the flavonoids have not supported their claims with data obtained from well controlled clinical studies. Until such studies are made, it must be concluded that the flavonoids are of little or no value in the treatment of disease.

By William N. Pearson, Ph.D.
Abstracted by Leonard M. Nevins, from J.A.M.A. Aug. 10, 1957, pgs. 1675-1677 (Council on Foods and Nutrition and Council on Drugs).

FLUORIDE OSTEOSCLEROSIS*

The authors report on 23 patients between the ages of 44 and 83 years with fluoride osteosclerosis whose roentgenographic records were collected from 170,000 roentgenographic examinations of the spine and pelvis of patients living in Texas and Oklahoma, where many communities have excessive fluoride content in their drinking water. The 23 patients had been drinking water with a fluoride content for many years of from 4 to 8 ppm. In each patient, adequate clinical examination failed to establish any relationship between the roentgenologic findings and the clinical diagnosis of the patient's condition. Each of the patients had lived his entire life in the same fluoride-bearing area in which he was born. An attempt was made to grade the osseous changes on the basis of a 1 to 4 scale, with grade 1 showing bone density of a very minimal degree and grade 4 showing bone density of an extreme degree. Pelvic ligament calcification was graded also on the basis of the 1 to 4 scale, with grade 1 representing minimal calcification in either the sacrotuberous or sacrospinous ligaments and grade 4 showing calcification extending about 6 cm. from the ischium toward the sacrum. Fifteen of the 23 patients had this type of ligamentous calcification, and it closely paralleled the degree of bone density.

The authors' roentgenographic experience with bone changes noted in the 23 patients confirmed Roholm's observation in that the earliest changes of fluoride osteosclerosis occurred in the pelvis and the lumbar spine. These changes consisted of slightly increased bone density, always evenly distributed throughout the vertebral bodies, and appearing bilaterally and symmetrically in the pelvis. There was a definite but slight "ground glass" appearance. The most advanced changes consisted of a chalky-white appearance of the vertebral column and pelvis and in addition a slightly increased density and coarse trabecular pattern in the ribs. Calcification of the sacrospinous and sacrotuberous ligaments began at the ligamentous attachment in the pelvis, usually bilaterally and extended towards the sacrum for distances up to 6 cm. The entire length of the ligaments did not calcify, and no instance of calcification at the sacral end of the ligaments was observed.

Because of the nature of this type of sclerotic process with associated involvement of the pelvic ligaments and the absence of any abdominal blood changes, the differential diagnosis of fluoride

osteosclerosis from other diseases producing increased bone density is not difficult. Fluoride osteosclerosis occurring in the patient exposed over a period of many years to fluorides as high as 8 ppm causes no harmful changes. Fluoride osteosclerosis is not evidenced roentgenographically in the patient drinking water with fluoride content of less than 4 ppm.

* Medical Literature Abstracts, J.A.M.A., Oct. 19, 1957, p. 904.
† By C. A. Stevenson and A. R. Watson. Am. J. Roetgenol. 78:13-18 (July) 1957 [Springfield, Ill.].

Book Reviews

Dental Clinics of North America-Symposium on Emergencies in Dental Practice, by James R. Cameron, D.D.S., Pages 333 to 617, with illustrations and cumulative index, W. B. Saunders, Philadelphia, July, 1957.

The title of this book is greatly misleading. One would expect to find an enumeration of the dental clinics on this continent and an evaluation of their services; instead it is a collection of articles by American authors, the majority of whom are from the eastern states.

The foreword and twenty-two chapters of this book, covering the management and the prevention of emergencies in the practice of general dentistry and oral surgery by a distinguished group of contributors are well written and to the point. Each subject is covered in approximately twelve pages. The photographs and photomicrographs add much to the subject matter. It would be impossible to single out any particular article. Suffice it to say that "Dental Clinics of North America" brings to the dental profession a new approach to the varied emergencies in the practice of general dentistry and oral surgery.

The chapters are divided into sections making for easier reading and most of the articles contain a summary. Not all have references.

The contributors include Leonard Monheim, Daniel J. Rossi, Ernest F. Ritsert, William E. Durbeck, James E. Chipps, Mario V. Troncelliti, Harry Seldin, S. Daniel Seldin, William Rakower, Sam F. Seely, Ogden M. Frank, Merle M. Miller, Raymond F. Huebsch, Donald E. Cooksey, John W. Hamilton, Stephen P. Mallett, Irving Meyer, Gerald Shklar, J. J. Stetzer, Jr., Lionel Gold, Sanford M. Moose, John H. Shackelford, Claude S. La Dow, Charles K. Kirby, Neal A. Harper, and John S. McQuade.

ANTHONY F. POSTERARO

Cortisone in Dentistry by Lyon T. Strean, M.Sc., Ph.D., D.D.S., F.A.P.H.A., Dental Items of Interest Publishing Co., Brooklyn, N.Y., 178 pages, 57 illustrations, 1957.

This monograph provides a medical basis for the application of adrenal cortical hormones in dentistry. Since this text is the first to be written on this subject for dentists the author has attempted and successfully so, to link the clinical observations in medical practice with their application in dentistry. Particular attention has been directed to those diseases which may complicate oral surgical procedures and which may lead to shock and death if necessary precautions are not taken. The recommendations made in this text may help save a life or aid in restoring health to those suffering from diseases amenable to adrenal cortical therapy.

Since the pituitary and adrenal glands are so important for the maintenance of body function, they are discussed in this text in detail. These glands and the hormones they secrete are of particular interest to the dentist because of their influence on oral inflammatory disease.

The author feels that it is difficult to separate those diseases which fall within the scope of dental practice and those of systemic origin requiring the attention of the physician. Physicians and dentists should coordinate their efforts and correlate diseases within the scope of dental practice and those of systemic origin.

This text is a welcome addition to the dental literature and can serve as a useful guide for both the practitioner and the specialist.

Living With All Your Heart in Health and Disease, by Henry C. Crossfield, M.D., F. A. C. P., Twayne Publishers, Inc., New York, 1957.

This is an important book for the layman written by a distinguished heart specialist. It presents all the things we should know about the nature and nurture of the heart in health and disease in a clear-cut manner. The book actually teaches how to maintain the well-being of the heart throughout life. After a very descriptive story on the makeup and workings of the heart, the author presents everyday methods of protecting the heart from irreparable damage. There is a detailed discussion of how the heart is examined by the doctor, the type of heart disease that plagues mankind and the effect of exertion, stress and strain on the heart musculature.

Dr. Crossfield indicates the need for every healthy person to know his own pulse rate when quiet and active, his own blood pressure, his electrocardiogram and chest x-ray film heart measurements. These are the facts the doctor determines of the diseased heart, hence the need for these norms for evaluating the degree of heart damage. There is excellent advice on the kind and type of exercise to improve the circulation, the type of diet to follow to prevent heart disease and some pertinent facts on the effect of alcohol and tobacco and other stimulants on the heart and blood vessels. The heart is pictured as a very tough muscle, perhaps, the strongest, if not the largest in the body. It can endure tremendous strains, but nervous strain may play havoc with the heart. The book is teeming with suggestions about guarding the heart with understanding and knowledge of its functions with a sensible diet, reasonable exercise and rest in order to get the greatest milage from one's heart. The book is well written, clearly illustrated, easily read and re-read to get the maximum helpfulness for one of the vital organs of the body.

I. NEWTON KUGELMASS, M.D.

Periodontia by Henry M. Goldman, D.M.D., F.A.C.D., and D. Walter Cohen, D.D.S., C. V. Mosby Co., St. Louis, 1957, ed. 4, $12.50.

In the preparation of the fourth edition of "Periodontia" the necessary revisions were far fewer than previously required. Much of the material from the third edition, exclusive of therapy, has been included together with new and pertinent information.

The basic problems of periodontia are discussed so that a concise picture of the examination procedure and diagnosis of the supporting and investing tissues can be appreciated. The authors have been very successful in their effort to correlate etiology and diagnosis with underlying tissue changes.

Of particular interest is the chapter devoted to the role played by bacteria in gingivitis and periodontitis. The authors point out that there is no clear cut evidence to indicate that bacteria are the primary factors responsible for the periodontal lesion. However, the bacteria are involved in the complex reaction which continues the gingival irritation. At the present time, research progress is being made to determine the mechanism of bacterial activity and histopathologic changes. The results of these investigations may present a more lucid picture.

The authors have compiled an excellent section devoted to periodontal disease in children. They demonstrate that the untreated periodontal lesion in the child will progress to further destruction in the adult. Doctors Goldman and Cohen indicate that any preventive program to combat periodontal disease will, of necessity involve the elimination of periodontal pathology in the child.

This text is devoted exclusively to the examination, causation and diagnosis of periodontal disease. No attempt has been made to discuss therapy. The details of treatment techniques form the basis for another text of which Dr. Goldman is co-author.

A History of Nutrition, Elmer V. McCollum, 423 pages, preface and indexed. Houghton Mifflin Company. Boston, 1957, $6.00.

The science of nutrition is presented here in its broadest perspective. Man's concept of foods from earliest times to contemporary are considered in chronological sequence. A fascinating panorama of human endeavor is painted, slowly, expertly, and meticulously, exploring the efforts that men have made to understand the relationship between ingestion, health and life.

Out of the welter of early superstition, taboo, experimental blunder and success, the historical accumulation of facts is reviewed and evaluated. In reading such a work one cannot but stand in awe at the magnitude of the importance of this subject, and at the rate at which it has been developing, especially in view of its recency as a science.

Early studies on carbohydrates, fats and proteins are presented in separate chapters. The discoveries on digestion are reviewed, as well as those on respiration and calorimetry. The relationship of modern chemistry and its evolution are well developed. The chapters on the physiological significance of microorganisms in the alimentary tract, and those on protein-rich and -poor diets were of particular interest to this reviewer.

The discoveries of the principal vitamins now known, in which the author had no small share, are well reviewed, in chronological sequence, as well as the roles of the fatty acids and inorganic constituents. The closing chapter on "new horizons" is both encouraging and rewarding.

This monument of ten years of historical research and digestion is a condensed meal. It should be tasted, savoured, consumed and enjoyed by all whom nutrition concerns to their advantage, dentist and physician, among others, included.

ARTHUR N. YOHAI

The Dentist and the Law. Charles Wendell Carnahan, Ph. B., J.D., LL.M., Jur. Sc.D., 263 pp, St. Louis, The C. V. Mosby Co., 1955, $5.25.

Carnahan has offered in a practical and concise manner a text based, on the case book method of law. Practicing dentists and students of dentistry will find this method more helpful in the understanding of legal questions usually found phrased only in highly technical language.

The 171 cases cited, together with an explanation of the basic rules of law involved, would normally take many volumes of a law library. The author has summarized many of the pertinent cases, particularly as they apply to the practice of dentistry, omitting

much that would be extraneous and burdensome. The book though brief, demonstrates clearly the importance and the need of basic knowledge of legal rules in the practice of dentistry. This brevity is of value to the busy practitioner.

The ten chapters of the book are subdivided into numerous sections. Many sections could have been grouped together under single headings, particularly since the book has an excellent index. Footnote citations are kept to a minimum, thereby eliminating many of the complexities found in legal volumes.

The author's experience as a teacher of Dental Jurisprudence has been invaluable in making this a very practical and handy book for the dentist's library.

ANTHONY F. POSTERARO

Dental Clinics in North America—Symposia on Tumors of the Oral Regions and Modern Practice in Endodontics. W. B. Saunders Co., Philadelphia. Pages 619 to 924, with illustrations and cumulative index, November 1957.

The first symposium is designed to give a general picture of the cancer problem. It reviews the oral neoplasms and their tumor-like counterparts region by region. This book gives some views on etiology and it discusses treatment by various methods and the dental role in post-therapy prosthesis. There are some points upon which the participants disagree slightly with each other, although all are thoroughly qualified and direct their discussions toward the ultimate goal of preserving human health and life.

The carefully treated root-filled tooth is no longer regarded as a menace to health. Unquestionably, the inadequate often careless techniques that were widely employed to treat pulp-involved teeth prior to the development of the concept of focal infection contributed to infection in the jaws. The consensus today is that demonstrable infection should not be allowed to remain in the body, but that the root-filled tooth that has been in the mouth for some time with no evidence of disturbance in the surrounding tissues does not jeopardize the health of its host.

There are 28 contributors to this symposia which thoroughly covers "Tumors of the Oral Regions" and "Modern Practice in Endodontics."

This text is an excellent guide for those practitioners and specialists who are interested in a modern and concise approach to the problems of "Tumors of the Oral Regions" and "Modern Practice in Endodontics." It is a welcome addition to the dental literature.

Academy Proceedings

NEW MEMBERS ELECTED AT MID-ANNUAL MEETING OF AMERICAN ACADEMY OF DENTAL MEDICINE — DEC. 8, 1957

Barron, John M.
Memphis, Tennessee

Camara, Jorge A.
New York, N. Y.

Cohen, Morris N.
Hartford, Conn.

Cummings, Paul M.
Chapel Hill, N. C.

Dickinson, James B.
E. Rutherford, N. J.

Gibilisco, Joseph A.
Rochester, Minn.

Green, Edward N.
Brooklyn, N. Y.

Greene, Robert L.
New York, N. Y.

Isenhower, Samuel H.
Newton, N. C.

Jarrett, Clyde H.
Charlotte, N. C.

Kendrick, Zebulon V.
Charlotte, N. C.

Kraus, Frederick W.
Birmingham, Ala.

Littman, Henry
New York, N. Y.

Martin, Harold G.
New York, N. Y.

Miller, I. Franklin
New York, N. Y.

Niditch, Irwin
Brooklyn, N. Y.

Rehm, Jerome G.
Charlotte, N. C.

Ross, Norman F.
Durham, N. C.

Sachs, Nathan J.
Jamaica, N. Y.

Sarnoff, Alvin M.
Jamaica, N. Y.

Sazima, Henry J.
Cleveland, Ohio

Stone, Samuel
Manchester, Conn.

Stroup, Paul A.
Charlotte, N. C.

Updegrave, William J.
Philadelphia, Pa.

Wallace, James C.
Chicago, Illinois

Wheless, John R.
Reidsville, N. C.

Zealy, James McKee
Goldsboro, N. C.

NEW JERSEY SECTION

The New Jersey Section of the American Academy of Dental Medicine held its first Clinical Meeting of the year on October 15th at the Hotel Douglas in Newark. The Clinician was Dr. Alexander Seelig, of Fairleigh Dickinson Dental School. His topic was "The Experimental Effects on the Pulps of Rhesus Monekeys with Histological Study Following the Use of Commonly Used Medicaments."

The meeting was well attended and the topic was presented in a most interesting manner evoking a considerable question and answer period.

The next meeting will be held February 17th, 1958 at which time we are co-sponsoring a joint meeting with the Essex County Dental Society. The clinician for that event will be Dr. Albert Schatz. His topic will be "The Proteolysis and Chelation Theory of Dental Caries."

The New Jersey Section has started preliminary work on the 1959 Annual Convention to be held in Atlantic City during May, 1959. The topics, speakers, location, and dates will all be announced in the near future. Reuben Feltman and Frank Schambach have been appointed co-chairmen of the convention and they are in the process of appointing their various committees.

The officers of the Section elected for the coming year are:

President ..... Louis B. Kelsten
President-elect ..... Reuben Feltman
Secretary-treasurer ..... Abraham I. Chasens
Editor ..... Morton DeScherer
Delegate ..... Ira F. Ross
Executive Board ..... Samuel Turkenkopf, Harold Lentz, Marvin Tuckman

At last year's convention in Boston, the following New Jersey men were accepted for membership:

Michael Frost, Henry J. Gewirtzman, Bernard W. Greenberg, Edward M. Grevatt, Alvin Hirschberg, Phillip Lowenstein, and Martin Protzel.

We are extremely proud of the caliber of these new members and feel that both the national and local units will be greatly benefited by their admission.

IRA F. ROSS, Delegate

AMERICAN ACADEMY OF DENTAL MEDICINE COMMITTEES—1957-1958

PUBLICATIONS

IRVING YUDKOFF, New York, *Chairman*	1958
LOUIS R. BURMAN, New York	1958
J. LEWIS BLASS, New York	1959
SIDNEY S. SORRIN, New York	1959
ALLAN N. ARVINS, New York	1960
SAMUEL C. MILLER, New York	1960

FELLOWSHIP & HONORARY MEMBERSHIP

DON C. LYONS, Jackson, Mich., *Chairman*	1958
GEORGE A. BRUNS, Boston	1958
HAROLD GELHAAR, New Jersey	1959
ARTHUR GOLD, Boston	1959
S. L. ROSENTHAL, Philadelphia	1960
GEORGE G. STEWART, Philadelphia	1960

CONSTITUTION

GEORGE A. BRUNS, Revere, Mass., *Chairman*	1958
GEORGE F. CLARK, Boston, *Co-Chairman*	1958
LOUIS R. BURMAN, New York	1958
JACK ENGLANDER, New York	1959
EDGAR JAMES, Detroit	1959
WALTER LEVINE, Baltimore	1960
ARTHUR GOLD, Boston	1960
Volunteer: HAROLD E. BRENNAN, New York	1958

PLANNING AND FINANCE

S. LEONARD ROSENTHAL, Philadelphia, *Chairman*
J. LEWIS BLASS, New York
GEORGE A. BRUNS, Boston
LOUIS R. BURMAN, New York
GEORGE F. CLARKE, Revere, Mass.
HAROLD GELHAAR, New Jersey
WILLIAM M. GREENHUT, New York
SAMUEL C. MILLER, New York
SIDNEY SORRIN, New York

MEMBERSHIP

ABRAHAM GOLDSTEIN, New York
and Representative of each section.

GRADUATE AWARDS AND ENDOWMENT

LAWRENCE STAPLES, Boston, *Chairman*	1958
JOSEPH NASSAU, Hartford	1958
EDWIN DEER, Michigan	1959
WALTER LEVINE, Baltimore	1959
GEORGE STEWART, Philadelphia	1960
ALEX KELTIE, Boston	1960

DENTO-MEDICAL RELATIONS

S. SIGMUND STAHL, New York, *Chairman*	1958
MORTON DESCHERER, New Jersey	1958
MAX HART, Michigan	1959
MAX GOLDENBERG, Montreal	1959
HORTON D. KIMBALL, Detroit	1960
EDWARD DOBBS, Baltimore	1960

HISTORIAN—William M. Greenhut, New York

SECTIONS

SAMUEL TURKENKOPF, Newark, *Chairman*	1958
ALFRED AUGUSTINE, Miami Beach	1958
MILTON SISKIN, Tennessee	1959
EDWARD T. FARRIS, Texas	1959
GUNTER SCHMIDT, St. Louis	1960
ROBERT DIAMOND, Boston	1960

ETHICS

J. LEWIS BLASS, New York, *Chairman*	1958
REUBEN FOGELSON, New York	1958
SIDNEY SORRIN, New York	1959
GEORGE F. CLARKE, Boston	1959
HAROLD GELHAAR, New Jersey	1960
HARRY ALBERT, New York	1960

and National Officers

MEMBERSHIP RETENTION

JOE BARKLEY, Detroit, *Chairman*	1958
SAMUEL ROBBINS, Ohio	1958
WILFRED J. JOHNSTON, Montreal	1959
EDWARD FARRIS, Texas	1959
TOLAR HAMBLEN, Texas	1960
EARL STONE, Boston	1960

KEYS

LOUIS R. BURMAN, New York, *Chairman* 1958
MARIE SALUTSKY, Detroit 1958
F. J. FABRIZIO, Washington, D. C. 1958
PHILIP LEVIN, California 1959
JANE C. O'MALLEY, Buffalo 1959
JOHN SHERMAN, Toronto 1960
ALONZO DEVANNA, New York 1960

EDUCATION

HAROLD E. BRENNAN, New York, *Chairman* 1958
DON MOORE, Ontario 1958
BERNARD SATUREN, Philadelphia 1959
GEORGE BRUNS, Boston 1959
GEORGE F. CLARKE, Revere, Mass. 1960
IRVING GLICKMAN, Boston 1960

RESIDENCY IN ORAL MEDICINE

SIDNEY SORRIN, New York, *Chairman*

DENTAL INFORMATION BUREAU

ANTHONY POSTERARO, New York, *Chairman*
MELVIN J. POMERANZ, New York, *Alternate*

MEMBERS OUTSIDE NORTH AMERICA

ARTHUR GOLD, Springfield, Mass., *Chairman* 1958
MAXWELL GOLDENBERG, Montreal 1958
LEON SUKIN, Philadelphia 1959
EDGAR JAMES, Detroit 1959
JAMES LADD, Pontiac 1960
GUY LEFRANCOIS, Montreal 1960

INSURANCE COMMITTEE

WILLIAM M. GREENHUT, New York, *Chairman* 1958
LOUIS R. BURMAN, New York 1958
SIDNEY SORRIN, New York 1959
HARRY ALBERT, New York 1959
JACK ENGLANDER, New York 1960
ALONZO DEVANNA, New York 1960

PROGRAM

DON C. LYONS, *General Chairman*
1958 Annual Meeting (Montreal)
GEORGE F. CLARKE, *National Chairman*
LOUIS J. ROSEN, *Local Chairman*
ALBERT LAFERRIERE, *Registration*

Dr. John Oppie McCall was presented with an Honorary Membership in the American Academy of Dental Medicine by Dr. Samuel Charles Miller at the Winter Meeting of the American Academy of Dental Medicine which was held on December 8, 1957 at the Hotel New Yorker.

AMERICAN BOARD OF ORAL MEDICINE

The number of certificates issued by the American Board of Oral Medicine now totals 48. Four members were certified at the examination given on May 30, 1957 in Boston.

The next examination will be given in Montreal on May 30, 1958. The Board is following the regular procedure for recognition by the Council on Dental Education in view of the fact that the moratorium on the recognition of dental specialties has now been lifted.

LESTER W. BURKET, *Secretary*

DR. SAMUEL CHARLES MILLER HONORED

Founder and First President of the Academy

Two hundred and fifty friends, colleagues and family of Dr. Samuel Charles Miller honored him at a testimonial dinner on Saturday evening, December 7, 1957 at the Hotel Roosevelt in New York City. The reception and dinner was tendered to him by the Periodontia and Oral Medicine Department of New York University College of Dentistry in celebration of his twenty-five years as Chairman of the Department.

Among those who paid tribute to Dr. Miller included Dr. James E. Aiguier, representing the American Academy of Periodontology; Dr. Marcel B. Archambault, President of the American Academy of Dental Medicine; Dr. Albert Epps, Dr. Miller's classmate; Past Presidents of the Academy, Sidney Sorrin, J. Lewis Blass and Louis R. Burman; Dr. Earl O. Butcher, Assistant Dean of the dental college, and Dr. Raymond J. Nagle, Dean of the dental college. Dr. A. W. Ward of San Francisco, a pioneer in the field of periodontia, extended to Dr. Miller his personal greetings and the best wishes of the California Academy of Periodontology.

Dinner Chairman and Toastmaster, Dr. Sidney Sorrin read many telegrams and letters of good wishes to Dr. Miller from friends all over the world. These greetings were incorporated into a book which was presented to the guest of honor. A beautiful portrait of Dr. Miller was unveiled by his two daughters, Barbara and Stephanie.

This tribute to Dr. Miller, which was so justly deserved, will be long remembered by those who were privileged to be present.

Dr. Samuel Charles Miller is recipient of an oil portrait of himself that was presented to him by Dr. Sidney Sorrin at a Testimonial Dinner tendered to Dr. Miller last December at the Hotel Roosevelt in New York City.

WINTER MEETING CLINICIANS

Howard A. Rusk, M.D. Arthur J. Barsky, M.D. Robin M. Rankow, M.D.

The Scientific Session of the Winter Meeting of the American Academy of Dental Medicine was an outstanding success. More than 175 members and friends heard three excellent speakers discuss the topic of "Rehabilitation".

The first speaker, Dr. Howard A. Rusk, Professor and Chairman, Department of Physical Medicine and Rehabilitation, New York University Bellevue Medical Center and Associate Editor, The New York Times, gave an excellent presentation on "Psychological Aspects of Rehabilitation". He was followed by Dr. Arthur J. Barsky, Attending Plastic Surgeon, Mount Sinai Hospital and Professor of Surgery, Albert Einstein College of Medicine, who spoke on "Rehabilitation Through Plastic Surgery". The last speaker of the program was Dr. Robin M. Rankow, Adjunct Surgeon, Montefiore Hospital; Assisting Attending Surgeon, Columbia Presbyterian Hospital; Assistant Professor, Columbia University and Consultant Editor, Head and Neck Cancer, New York State Dental Journal, who presented an excellent paper on "Immediate and Delayed Rehabilitation of the Head and Neck Cancer Patient."

Dr. John Oppie McCall, noted author and clinician, was awarded an Honorary Membership in the American Academy of Dental Medicine. This presentation was made by Dr. Samuel Charles Miller on behalf of the Academy.

Special mention should be given to Dr. Sheldon J. Ross, Chairman, and to Dr. Nathan Wachtel, Co-Chairman and their Committee for the excellent manner in which the Winter Meeting was conducted. These stimulating programs will insure even larger audiences in the future.

TWELFTH ANNUAL CONVENTION

MONTREAL, QUE. . MOUNT ROYAL HOTEL

MAY 29, 30, 31, 1958

ACADEMY MEMBERS IN THE NEWS

Academy Past President, Dr. S. Leonard Rosenthal visited Chile and Argentina last July and August. From July 8-17th he conducted a postgraduate course on Oral Medicine at the University of Chile School of Dentistry in Santiago. At the final session, he was made an honorary member of La Sociedad Odontologica de Chile and La Sociedad Chilena de Paradentologia. On August 1st, Dr. Rosenthal addressed the Department of Dermatology and La Sociedad de Estomatologia at Rawson Hospital, Buenos Aires, on "Oral Dermatoses". On August 3rd he was guest speaker at a faculty meeting at the Dental School, University of Buenos Aires. The subject was: "The Course in Oral Medicine at Temple University".

Dr. S. Leonard Rosenthal was presented with an Honorary Membership in the Chilean Dental Association last summer during his lecture tour of several South American countries.

Dr. Samuel Turkenkopf was invited to lecture on Endodontia at the F. D. I. Dental Congress in Rome last September. While in Europe he visited the Norwegian Institute of Dental Research in Oslo, Norway.

Dr. Lawrence Staples has been giving postgraduate courses in hypnosis technique at Tufts University School of Dental Medicine. He recently took part in a coast-to-coast telephone hook-up which marked the organization of the American Society of Clinical Hypnosis with dentists, physicians and psychologists as members.

Dr. Don C. Lyons and his wife toured Europe last summer and visited Scotland, England, Italy, Switzerland and France. Dr. Lyons presented a paper entitled "Use of Cortisone Preparations in the Treatment of Dental Pain" at the International Dental Congress in Rome. His talk was illustrated with kodachrome slides and demonstrated the effects of this method in the temporary control of pain until complete diagnosis and more permanent methods are instituted.

Dr. George G. Stewart and his wife were guests of the American Dental Society of Europe at its annual meeting which was held last August in Lausanne, Switzerland. Dr. Stewart presented a clinic on research involving an evaluation of root canal sealing agents and introduced a new polyketone substance for this purpose. He also presented a new film "Surgical Endodontics" that he produced. This film is in color with sound and animation.

Dr. Ed Freimuth, Dr. Edwin Deer and Dr. Horton D. Kimball, all members of the Michigan Section of the Academy, presented a symposium on Dental Medicine at the 16th Annual Dental Review of the Detroit District Dental Society last November. This presentation gave the Michigan Section a great deal of prestige in the Detroit area.

Heartiest congratulations go to academy members Leonard Nevins and Sigmund Stahl who recently passed the Board of Oral Medicine, and to Marvin Okun, Irving Yudkoff, Theodore Lite and Sanford Kirsch who passed the examinations for the Board of Periodontology.

* * * * * *

Section Presidents: Send news items about your members to the editor.

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Samuel Charles Miller and Staff:

No. 124. REFRESHER COURSE IN PERIODONTIA AND ORAL MEDICINE

Ten sessions, Wednesdays, 4:00 to 6:00 P.M. February 12, 1958 to April 23, 1958.
Tuition and books. $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occlusion is performed by the student on casts. Patient demonstrations include equilibration of occlusion, conservative and radical therapy, home care, etc.

No. 123. PERIODONTIA AND ORAL MEDICINE IN THE SPANISH LANGUAGE

Four weeks, Monday through Friday, 9:00 A.M. to 4:00 P.M.
March 3 to March 28, 1958 inclusive
Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

No. 122. COMPREHENSIVE PREPARATION IN PERIODONTIA AND ORAL MEDICINE

No. 122 A. One academic year, full time. September 17, 1958 to May 22, 1959.
Tuition $1,010.00. Instruments and books approximately $135.-00.

No. 122 B. Two academic years, full time. September 17, 1958 to May 27, 1960.
Tuition $1,770.00 (two years). Instruments and books approximately $231.00.

No. 122 C. Two academic years, half time. September 17, 1958 to May 27, 1960. (Two and one half days weekly.)
Tuition $1,010.00 (two years). Instruments and books approximately $135.00.

No. 122 D. Four academic years, half time. September 17, 1958, to May 25, 1962. (Two and one half days weekly.)
Tuition $1,770.00 (four years). Instruments and books approximately $231.00.

These courses are designed for comprehensive preparation in Periodontia and Oral Medicine and are of particular significance for certification towards specialization. All didactic and clinical phases are covered with the objective of developing expert proficiency.

No. 222. GRADUATE COURSE IN PERIODONTIA AND ORAL MEDICINE

Two academic years, full or half time, beginning Sept. 17, 1958. Tuition $2,020.00. (two years). Instruments and books approximately $275.00.

This program leads to the degree of Master of Science (in Dentistry). The objectives are to teach superior proficiency in Periodontia and Oral Medicine based on adequate background in the basic sciences.

DOCTORS' ORCHESTRAS

Dentists and members of allied professions are invited to become members of two doctors' orchestras in New York City. Non-medical musicians, may also join. The Doctors' Orchestral Society of New York rehearses Thursday evenings in the auditorium of the Stuyvesant High School, 15th Street and First Avenue, New York City at 8:30 P.M. The Brooklyn Doctors' Symphony meets Wednesday evenings in the auditorium of the Brooklyn High School of Homemaking at 901 Classon Avenue, corner of Washington Avenue, Brooklyn at the same time. Both groups are rehearsing now.

For further details contact: Dr. Benjamin A. Rosenberg, 909 President Street, Brooklyn, New York, NE 8-2370.

TEMPLE UNIVERSITY DENTAL SCHOOL POSTGRADUATE COURSES JANUARY-JUNE 1958

Full Denture Construction	Dr. Bernard Jankelson	January 6-10, 1958
Advanced Denture Construction	Dr. Bernard Jankelson	January 13-18
Advanced Orthodontics	Dr. Robert H. W. Strang	January 19-February 1
Periodontal Prosthesis	Dr. Morton Amsterdam Dr. D. Walter Cohen	February 10-14
Practice Administration	Dr. Jay H. Eshleman	February 15
Practical Uses of Electrosurgery in Dentistry	Dr. Maurice Oringer Dr. Martin Entine	March 1
Oral Medicine	Dr. Leonard Rosenthal Dr. Lester Burket	March 3-7
Advanced Surgical Technique in Periodontal Therapy	Dr. Saul Schluger	April 21-25
The Washed Field Technic in Accelerated Dentistry	Dr. E. O. Thompson	May 5-6
Endodontics—Immediate Root Resection	Dr. Louis Grossman Dr. Leonard Parris	June 3-8
The Porcelain Jacket Crown	Dr. Joseph Ewing Dr. M. J. Kotanchik	June 11-12 18-19, 25

For complete details concerning these courses, all of limited enrollment, please write to: Postgraduate Division, School of Dentistry, Temple University, Broad above Allegheny, Philadelphia 40, Penna.

BETH ISRAEL HOSPITAL BOSTON 15, MASSACHUSETTS

The Dental Department of the Beth Israel Hospital announces a postgraduate course in Periodontia to be given in the Spring of 1958.

A two-week seminar in Periodontia is scheduled for March 17 to March 28. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations, and clinics. The tuition fee is $350.00. A deposit of $150.00 is necessary for enrollment.

Co-Directors of the course are Henry M. Goldman, D.M.D. and Bernard Chaikin, D.M.D.

For further information, write to:

Director of Public Relations and Education, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Massachusetts.

THE UNIVERSITY OF ALABAMA SCHOOL OF DENTISTRY

The following refresher courses will be offered by the University of Alabama School of Dentistry in Birmingham, Alabama during the months of February, March and April, 1958. Further information about these courses may be obtained by writing to the Director; Refresher Course Program, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

Periodontology for the General Practitioner will be given by Sumter S. Arnim, D.D.S., Ph.D., February 8, 9, 10, 1958.

Preventive Orthodontics will be conducted by Earl E. Shepard, D.D.S., February 15, 16, 17, 1958.

Laboratory Procedures in Crown and Bridge Prosthesis for the General Practitioner will be under the direction of William D. Powell, D.M.D., February 22, 23, 24, 1958.

Occlusal Adjustment: Its relation to dental, periodontal and temporomandibular joint problems will be given by Sigurd Ramfjord, L.D.S., M.S., Ph.D., March 1, 2, 3, 1958.

Applied Maxillofacial Anatomy will be conducted by Harry H. Shapiro, D.M.D., March 8, 9, 10, 1958.

Practical Exodontia will be given by Daniel J. Holland, D.M.D., March 29, 30, 31, 1958.

Root Canal Therapy will be under the direction of Floyd D. Ostrander, D.D.S., M.S., April 12, 13, 14, 1958.

Pedodontics for the General Practitioner will be conducted by Sidney B. Finn, D.M.D., M.S., April 19, 20, 21, 1958.

TWENTY FIFTH ANNIVERSARY MEETING OF THE NEW YORK INSTITUTE OF CLINICAL ORAL PATHOLOGY, INC.

At The New York Academy of Medicine Building

2 East 103rd Street, N.Y.C., Room 440

Friday, January 24, 1958

PROGRAM

10:00-10:30 A.M.—History of Oral Pathology.

Lester R. Cahn, D.D.S., F.D.S., RCS (Eng. Edin.), Associate Professor of Oral Pathology, Faculty of Medicine, Columbia University; President of the New York Institute of Clinical Oral Pathology.

10:35-11:05 A.M.—The Dentist's Role in the Diagnosis of Lesions of the Oral Mucosa.

Hamilton Burroughs Greaves Robinson, D.D.S., M.S., Associate Dean, Professor of Dentistry, Ohio State University.

11:10-11:40 A.M.—Salivary Gland Disease.

David Weisberger, D.M.D., M.D., Professor of Dental Medicine, Harvard School of Dental Medicine.

11:45-12:15 Noon—The Importance of Histologic Study of the Various Radioluscent Areas of the Jaws.

Charles A. Waldron, D.D.S., M.S.D., Professor of Pathology, Emory University School of Dentistry.

2:00-2:30 P.M.—New Concepts of the Supporting Dental Structures and Periodontal Disease.

Harry Sicher, M.D., D.Sc., Professor and Head of the Department of Anatomy, Loyola University School of Dentistry, Chicago College of Dental Surgery.

2:35-5:00 P.M.—Clinical Oral Pathology Conference.

Joseph L. Bernier, Colonel, Dental Corps, U.S. Army, F.D.S., RCS (Eng.), Registrar and Pathologist, The Registry of Oral Pathology, Armed Forces Institute of Pathology.

Robert B. Shira, Colonel, Dental Corps, U.S. Army, Chief, Oral Surgery Section, Walter Reed Army Hospital.

Lester R. Cahn.

This meeting is open to the Dental, Medical and Allied Professions.

Please advise as soon as possible whether you will attend, so that seating facilities may be arranged, by communicating with the Executive Secretary, The New York Institute of Clinical Oral Pathology, 101 East 79th Street, New York 21, N. Y.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

The following courses listed below will be presented at Tufts University School of Dental Medicine during 1958. Application and further information may be obtained by writing the Director, Division of Graduate and Postgraduate Studies. Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Mass.

DPG. 603—PERIODONTICS

Wednesday, Thursday, Friday—9 A.M.—5 P.M.
February 26, 27, 28, 1958

Tuition $90.00 — Enrollment limited

Dr. Balint Orban, Professor of Periodontia, Loyola University, Chicago, Illinois

DPG. 606—DIAGNOSIS AND MANAGEMENT OF OCCLUSAL DISHARMONIES

Monday, Tuesday, and Wednesday
April 7, 8, 9, 1958

Tuition $75.00 — Class limited to 12

Dr. W. George Schmidt, Chicago, Illinois

DPG. 703—CROWN AND BRIDGE PROSTHESIS PARTICIPATION COURSE

For Eight Days—9 A.M.-5 P.M.
On the following dates
February 28, March 1, 7, 8, 14, 15, 22, 29, 1958

Tuition $200.00 — Class limited to eight

Dr. R. Sheldon Stein, Boston, Massachusetts

UNIVERSITY OF MINNESOTA SCHOOL OF DENTISTRY

The Dental and Medical Faculty of the University of Minnesota will present a course in Advanced Oral Pathology during the week of April 28 through May 2, 1958. Both lectures and pathologic slide study will be included. Microscopes will be provided. The material presented will include: neoplastic and non-neoplastic diseases of the salivary glands, neoplasms of dental origin, bone pathology, soft tissue neoplasms, dermatologic pathology, diseases of the lymphoid system, and oral roentgenographic evidence of systemic disease.

The faculty will include Dr. Robert Lukes, Armed Forces Institute of Pathology, Dr. D. C. Dahlin and Dr. Edward Stafne of Mayo Clinic, Dr. Robert Goltz of the Medical School, University of Minnesota, Dr. Anand Chaudhry and Dr. Robert J. Gorlin of the School of Dentistry, University of Minnesota.

The tuition will be $50 for the week. If room and board at the Center for Continuation Studies are desired, the cost for the week will be about $50.

Interested individuals will please contact Dr. Robert J. Gorlin, Chairman, Division of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis, Minnesota, for further information.

Voici!

TWELFTH ANNUAL CONVENTION

AMERICAN ACADEMY OF DENTAL MEDICINE

MONTREAL, QUE. MOUNT ROYAL HOTEL

MAY 29, 30, 31, 1958

◂ • ▸

Excellent Ladies Program — Reception — Dance

DISTRIBUTOR

PERIODONTAL INSTRUMENTS
and MEDICAMENTS

for

PERIODONTAL CLINICS

and

POST GRADUATE COURSES

GUTERMAN DENTAL SUPPLY CO.

515 MADISON AVE. • NEW YORK 22, N. Y.

COLLEGE BRANCH

203 East 23rd Street • New York 10, N. Y.

Phone: PLaza 9-5020